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Disease, Environmental, and Management Factors Related to Poultry Health

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PREFACE

This publication contains the papers presented at a symposium on poultry health sponsored by the Agricultural Research Service, U.S.D.A., and the University of Georgia, held at Jefferson Auditorium, U.S.D.A., Washington, D.C., March 20-22, 1961. Farm Research Divisions concerned with the planning and programming of this meeting were Animal Disease and Parasite Research, Agricultural Engineering Research, and Animal Husbandry Research. Some 250 scientists attended, representing Federal research and regulatory agencies, state agricultural experiment stations, and private industry.

Symposium speakers discussed new developments in disease, environmental, and management factors related to poultry health; the responsibility of Federal and State agencies and industry in reducing condemnation losses, and the potentials for solving current problems in this field through the joint efforts of scientists, regulatory workers, and the poultry industry.

It is hoped that both the symposium and the publication of the presentation papers will contribute to a more rapid advancement in research on those factors related to poultry health and to an early resolution of many condemnation problems.

Issued October 1961

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A NEW IMAGE FOR AGRICULTURE

By Frank J. Welch[1]

The groups represented in this audience serve an industry that is a striking example of how the whole economy benefits when technology is put to work in agriculture.

Improvements in poultry breeding, feeding, disease control, management, and marketing have brought nutritious foods, which were once a luxury, within the reach of most of our people.

The United States has contrived the best system of agricultural research and education the world has ever seen. Our goal is to maintain and strengthen this system. Our success in meeting that goal depends on public support.

A barrier that must be overcome is a public image of agriculture, a false image now widely held by people who have little or no first-hand knowledge of agriculture and its problems.

The number of people who have only meager or skewed or misleading knowledge is increasing. According to the 1959 agricultural census, those who don't live on farms now outnumber those who do by more than nine to one.

The estimated farm population of April 1960--15,635,000--was about the same as the farm population a hundred years ago when half of the people in this country were on farms.

The public image of agriculture has been distorted by too much stress on problems created by surpluses and by too little information about the benefits of abundance.

Five fallacies about agriculture have gained widespread currency:

The first fallacy is that food costs are increasing much faster than other living costs and are responsible for inflation. The fact is that U.S. consumers pay about the same share of their income for food (21 to 23 percent) as in the years immediately before World War II. And, as a matter of fact, if they bought the same food basket today as 20 years ago--in terms of variety and quality--only 16 percent of average consumer incomes would go for food.

Rent, transportation, and medical care have gone up twice as much as food in the past 10 years. Food costs have just about kept pace with the general price rise. Both have doubled since 1939. The average American income during that period has increased fourfold.

The second fallacy is that the farmer is chiefly responsible for the cost of food. He gets the lion's share of the food dollar. What should be made clear is that on the average, the farmer doesn't even get half of the food dollar. His share varies by commodity. On the average, in 1960, it was only 39 cents.

A third fallacy is that farmers are getting rich at the expense of the rest of us. The fact is that the average per capita income of the farm population from all sources in 1960 was less than a thousand dollars ($986). Average per capita income of the nonfarm population was two and one-third times as great. Total net income for all farms in the United States averaged $2,568 in 1960--almost $500 below the record high of 1948.

Costs and returns vary by types of farm. USDA estimates costs and returns on 32 important types of commercial farms in major farming areas of the country.

Farm income (net) rose on 13 types of farms, declined on 17 types of farms, remained the same on one type (Kentucky tobacco-livestock farms) during the 1950's as compared with 1947-49.

The highest losses reported in this series were on typical egg-producing

[1] Assistant Secretary, U. S. Department of Agriculture.

farms in New Jersey. Average net farm income between 1949 and 1958 was $3,600, 40 percent below the 1947-49 average. And in 1959, New Jersey egg producers operated at an average loss of $1,335.

Earnings have declined despite a steady increase in the size of the operation and a 10 percent rise in production. These improvements were not enough to offset rising production costs.

A fourth widely accepted fallacy is that farm support programs are wasteful. They encourage the build-up of surpluses. What is not recognized is that support programs are necessary to protect our investments in agriculture. We have built an agricultural plant without historic parallel in the abundance and diversity of its output. We have built an agricultural plant that produces nearly two-thirds of all the raw materials used by the American economy and that ranks with the best anywhere in the efficient use of land and labor.

Between 92 and 95 percent of our agricultural abundance moves through regular marketing channels. Production is fairly closely adjusted to demand for livestock products and fruits and vegetables. Our surpluses are chiefly in wheat, feed grains, and cotton--storable crops that, if necessary, can be used to meet emergencies at home or in other parts of the world. Bold, realistic support programs now in prospect will help alleviate the problem of surpluses.

The fifth fallacy is that the cheapest way to solve the problem of surpluses is to make drastic cuts in farm research. The fact is that research across the whole scientific spectrum is required if our farmers are to protect gains already made, keep in step with changing times, and sell on new markets.

Research in livestock production has not yet provided the farmer with lines of animals and techniques for getting full crops of chicks, poults, lambs, calves, and pigs. The losses--now averaging about 30 percent--represent an important cost. We have just begun to study the use of hormones, the influence of day-length, and other tools that may one day make it possible to assure full crops of young animals at any season.

Livestock breeders are just beginning to search for germ plasm that carries resistance to parasites and diseases--a genetic tool that has been invaluable in crop improvement.

A striking gain in this work is the discovery that certain lines of laying hens carry resistance to the avian leukosis complex. We may be able to control this virally transmitted disease through breeding.

We know very little about the nature of resistance to pests of both livestock and crops. Basic research on all phases of immunology is an urgent need.

We must speed up our research on the metabolism of crop plants and livestock species, on their parasites, predators, and competing species. We must know completely their chemistry, genetics, nutrition, and ecology. There are critical questions to be answered across the board. The questions call for research of the most fundamental sort.

We have by no means realized the full promise of the chemical age of agriculture. Chemicals now in use and those in prospect have created an urgent need for more research on the action of pesticides and their fate in plants, animals, and soils. We should know enough about the behavior of every agricultural chemical to construct a balance sheet that accounts for the chemical from application until its ultimate fate is known.

The high cost of feeding experiments, both in time and money, is well known. It is a serious obstacle to rapid progress in improving the nutritive value of food and feeds. This is one of the challenges to agricultural chemistry. Its solution is a vital step toward improvements in nutrition.

An influence that will shape farm markets in the years ahead--as it has in the immediate past--is the growing knowledge of nutrition--knowledge of the composition of foods and man's need for different foods for health at various stages of life.

Nutrition research in USDA and the State experiment stations on the food needs of healthy individuals is a valuable supplement to the information on nutritional diseases now coming out of medical research.

Recent findings in nutrition research suggest relationships not previously recognized between food and health. Intensive work will be required to give us a better understanding of the role of fats in our diets and to answer many other complex questions now being raised.

Farm programs must be revised in light of advancing technology, promise of new markets, and changing foreign

policies. Realistic programs must be backstopped by research throughout agricultural science.

More economic information is vital to sound adjustment. There is a pressing need also to expand significantly research that will give us guides for rural development programs. National interest requires that research find ways to improve incomes of those who suffer hardships as a result of technical and other changes beyond their control.

Our Nation has built an agricultural technology that is without parallel in diversity and efficiency of production. We have devised a unique system of research and education for maintaining that technology and for retooling it to keep pace with changes in other sectors of the economy. U.S. agriculture faces many crucial problems. Our success in meeting them depends on full understanding and support of this work by the public.

WELCOME

By Byron T. Shaw[1]

It is a pleasure to welcome you here to the U.S. Department of Agriculture and to this symposium on poultry health being sponsored in cooperation with the University of Georgia.

The Department has long had an interest in agricultural research. From its founding in 1862, up until the turn of the century, nearly three-fourths of all the money spent by USDA was spent on research. So, in effect, we started out as a research institution, and research has continued to be an important function.

The Nation has benefited tremendously from this foresight. In the hundred years since the Department of Agriculture was created we have spent--in all--$1.8 billion for research. If you add to this, the $1.4 billion that the States have spent, and an estimated $2.6 billion that industry has spent, you have a total of $5.8 billion that have been spent on agricultural research in this country.

This has been an extremely small investment when you consider how handsomely it has paid off. For example, if farmers today were still using the practices that were available to them just 20 years ago, it would cost us 13 billion dollars more a year to produce our food and fiber. And when you consider the additional benefits from research in the utilization, marketing, and home economics fields, it is certainly conservative to say that the Nation is getting a return every year of more than twice as much as the total cost of all agricultural research in the last 100 years . . . a more than generous return on our research investment.

The past 20 years alone have seen a remarkable growth in productivity and efficiency on our farms. One of the most dramatic success stories can be seen in the broiler industry, where there has been spectacular growth and development all the way round.

But such advances bring their problems too. For example, the close confinement of poultry . . . so essential to efficiency . . . has brought with it new problems of disease and disease management . . . problems of such size and complexity that they have brought together in this symposium the best minds from industry, the States, and USDA to lay the groundwork for solutions.

Livestock and poultry diseases of all kinds cost the Nation a lot of money. So much money, in fact, that the problem deserves a more intensive attack than we have made in the past. Fortunately, from our standpoint in the Department, we are now getting the facilities we need to move ahead on these diseases.

The establishment of the Plum Island Animal Disease Laboratory, New York, in 1956 was an important step in an intensified research program to help us fight animal diseases with all of the skill, imagination, and thoroughness that our scientists have at their command. Plum Island, as you know, is devoted to the study of foreign livestock diseases and is an extremely important outpost in our strategy of being forearmed and ready to deal with any foreign disease that might turn up in this country.

The new National Animal Disease Laboratory at Ames, Iowa, devoted to the study of domestic diseases is now about ready to move in to. It is a fine laboratory, it will have an excellent staff, and we will begin to see now the stepping up of much-needed research on our domestic diseases. One of the first laboratory units to be activated at Ames will be the one dealing with chronic respiratory disease.

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[1] Administrator, Agricultural Research Service, U. S. Department of Agriculture.

With the Ames laboratory in operation, facilities will be available at Beltsville for a badly needed expansion in research on parasites and parasitic diseases of livestock and poultry.

Of special interest to you, of course, are the two new poultry laboratories which Congress provided for only last year. These new facilities are designed specifically for research on the inter-relations of disease, environment, and management, under relatively controlled conditions. The diseases to be studied are those causing the highest condemnation losses in dressed poultry in the Southeast.

Construction of the larger of the two laboratories--the one on the University of Georgia campus--will begin in July. This new laboratory is to be organized around an inter-disciplinary approach--several research divisions working on a common disease problem. Sixty percent of the work will be devoted to disease research, with the other 40 percent divided between management and engineering. But in both these areas, the work will be pointed toward disease problems. The bio-engineering work, for example, will relate building materials, construction methods, temperature, and humidity to poultry disease.

The laboratory at State College, Miss., will place primary emphasis on management and engineering. Scientists there will work with commercial-size units of broilers to study the influence of environment and management on condemnation losses. The investigations to be made are those desired by broiler growers and the State Experiment Stations.

The work of these two new poultry laboratories will, of course, be closely coordinated with other Department work on poultry disease as well as with the research that is being conducted by the State Experiment Stations.

In the next 3 days you people will have an exceptional opportunity to exchange knowledge about the latest research on poultry diseases, the problems poultry producers face, and the proposals for solving these problems. The increasing importance of poultry meats and other products in the Nation's food supply is motivation enough to set your sights on the highest standards of poultry health. We will look forward to the contributions that this symposium will make.

If there is anything you would like to see, or anyone you would like to contact during your stay, I hope you will call on us. Best wishes for a stimulating, productive meeting.

SECTION I--RESPONSIBILITY OF THE FEDERAL AND STATE AGENCIES AND INDUSTRY

THE ROLE OF RESEARCH IN SOLVING THE POULTRY CONDEMNATION PROBLEM

By T. C. Byerly[1]

The first step in research, at least in problem-oriented or so-called applied research, is to define the problem. One of the purposes of this symposium is definition of the problem or problems to be solved, in order to develop methods for reducing condemnation losses in broilers. Each participant will contribute to that definition. From the standpoint of economics, 2 percent condemnation loss represents about 0.4 cent per pound cost on about 5 billion pounds of broiler produced, or 20 million dollars. To this should be added another 15 million dollars expended for drugs, immunizations, and other treatments. Add to these costs at least a cent a pound for extra feed required to bring our total broiler flock to market because growth was retarded by diseases, parasites, and environmental stresses--another 50 million dollars. An annual cost of 85 million dollars in all. Your estimate may differ from mine; but it will be very large.

To the parasites, diseases, and environmental stresses must be added genetic deficiencies of the broilers grown. Every broiler flock is a competitive society--competitive for food, water, space; competitive against parasites, diseases, and environmental hazards. Genetic capacity is a factor in competition.

Obviously, the problem of condemnation losses is a complex problem. Its solution requires research in many scientific disciplines. Basic research and applied research are both needed. We know that there are three pathways to better methods for producing broilers of better quality with minimal condemnation losses. We shall need to do research on all three: On environmental control to provide better growing conditions in the absence, in the prevention of transmission, and in the presence, of disease; on control of disease through immunization, genetic resistance, prophylaxis, and therapy; and finally, on disease eradication. Research in these three areas should be concurrent and coordinated. The construction of two new laboratories at the University of Georgia and Mississippi State University will provide a place for a few people to work, but will in nowise substitute for the many others already doing research on the problem at State experiment stations, in industry laboratories, and other research institutions. Among the 24 laboratory units soon to be activated at the National Animal Disease Laboratory at Ames, one of the first to be activated will be used for the study of chronic respiratory disease in poultry.

Research on so broad a problem, by research workers in many disciplines, will be benefitted by team research. Teams will be drawn from among the pathologists, nutritionists, geneticists, parasitologists, chemists, immunologists, engineers, physicists, biometricians, behavioral scientists, and poultry husbandmen working on the problem. Effective

[1] Deputy Administrator, Agricultural Research Service, U. S. Department of Agriculture.

teams are made up of competent people, each working effectively in the area of his own competence, exchanging information and ideas, sometimes working with a common pool of experimental animals and facilities, sometimes with a recognized research leader, but always planning together, encouraging and criticizing one another, often griping and grumbling, but working.

The role of research is affected by its objectives. In general terms, research will be designed and conducted to reduce condemnation losses among broilers. But research will provide only the means and the knowledge. Education and action are necessary to put research results to work. Some of you will choose as your objective the development and improvement of methods for the eradication of disease, including, of course, parasitic disease and metabolic disease. Others will define the problem in terms of protection against disease, the development and improvement of methods of disease control. Each individual, each team undertaking research intended to contribute to the ultimate solution of the broad problem will set more immediate objectives. Let us consider a few of these.

First, let us consider the basic research problem of the relation between host and parasite. Whether the parasite be viral, fungal, bacterial, protozoan, helminthic, or arthropodal, whether it be obligate or facultative, this relationship between host and parasite is one that must be studied as a part of research to solve the problem of condemnation losses in broilers.

Brilliant and dramatic recent discoveries have kindled great enthusiasm for research in this area. Deoxyribonucleic acid (DNA) has been demonstrated to be the structural material by means of which genetic information is transmitted from generation to generation. Is such information also transmitted from host to parasite, or vice versa? "DNA makes RNA [Ribonucleic acid], RNA makes protein" has been described as the ditty of the graduate student.

A recent paper by Work (1960) contains the following statement:

> "It is known from the work of Hershey that, when E. coli is infected with the bacteriophage T2, the immediate reaction is the injection of the phage DNA into the bacterium. Volkin (1958) has shown that this is followed by a period of vigorous RNA synthesis. During this period of rapid RNA turnover the newly formed RNA can be readily labeled by P^{32} or by C^{14}. The ratio of the specific activities of the bases (probably a reflection of base composition) in this RNA was cytidilic 18, adenylic 32, urydilic 32, guanylic 18. This is strikingly similar to the molar base composition of E. coli DNA or from the specific activity distribution in RNA obtained from control, uninfected E. coli cultures. These results suggest that viral DNA provides a template for the synthesis of a new type of RNA. A scheme in which RNA synthesis and RNA base sequence are dependent upon a combined RNA-DNA template has other attractions, which are too speculative to justify full development here; suffice it to say that such a scheme could be used to explain antibody synthesis, differentiation, and possible enzyme induction."

Many viruses have no DNA, just as many parasitic helminths have no gut; they are obligate parasites. Do they use host DNA as a tool in self-propagation?

Must obligate parasitic viruses that have only RNA find host cells with genetically compatible DNA to which the viral RNA may attach itself in order to reproduce? Can research find ways of inducing or enhancing host incompatibility? Of course, it already has, by provoking the host mechanism to produce antibodies by inoculation with appropriate antigens; but not all hosts are easily provoked. Must we breed for genetic resistance to disease, as Fred Hutt may argue and plant pathologists generally accept and practice?

Conversely, what can we do about the apparent capacity of parasite to accommodate itself through mutation? Insects resistant to pesticides have become too commonplace. Bacteria have developed resistance to sulfas. The facility of the common staphylococcus, Micrococcus aureus, to thrive in the presence of many broad-spectrum antibiotics is well-known. It is no comfort to learn that deaths from infection in man are increasing just as broiler condemnation losses seem to be. Dauer (1961) reported that mortality from all types of septicemia has been increasing steadily since 1948, particularly septicemia caused by staphylococci.

Research on defense mechanisms of host and parasite and of their enhancement by genetic chemical or allergic means is only one of the alternate research pathways available. The road to establishment of gnotobiotic or specific pathogen-free stocks is also available. Certainly such stocks are much to be desired as research materials. Is it really possible to free a poultry population of all viruses? Certainly it would be cheaper in the long run for poultry producers if poultry diseases and parasites were eradicated in the flocks of the U.S.A. We have progressed a long way toward realizing such a goal for (Salmonella pullorum. But we aren't even trying to eradicate coccidia. We once did try. Should we try again? Eradication of even one disease organism from all our poultry flocks would cost millions of dollars. But suppose our flocks were free of Newcastle, as we suppose they were prior to the thirties, we'd save a lot of vaccination cost, a lot of condemnation losses, and gain some export markets.

Would development of gnotobiotic and specific pathogen-free stocks render them catastrophically vulnerable? Certainly first exposure of coccidiosis-susceptible stock to a heavy infectious dose is so often disastrous that we don't take the risk but try to protect our broilers against certain exposure by early moderate exposure and medication at critical times. Would gnotobiotic stocks succumb to bacteriemia upon exposure under environmental stress? Certainly such stocks would be hatched with no egg-transmitted, passive immunity.

There is a belief widespread among laymen that a population reared in the absence of disease may be overwhelmingly susceptible to that disease. And indeed it may be. Early exposure to anaplasmosis or coccidiosis in low dosage does permit immunity to develop. But there is a more important reason; passive immunity, egg-transmitted, uterine-transmitted, or colostrum-transmitted is a principal means of protecting the neonatal young against infections until they develop the capacity to produce antibodies in effective amounts. This passive immunity has nothing directly to do with genetic resistance, though it has sometimes been interpreted as genetic.

The relationship between host and parasite is, of course, conditioned by genes. Achievements of plant breeders in producing new cereal breeding stocks resistant to rust races is phenomenal, but apparently never-ending. The skill of the plant breeder is matched by the capacity of the rust to change. Research by Flor (1956) has given a solid base for understanding the relationship of host and parasite in flax rust and flax. He found that if a host plant has a particular gene for rust resistance, it could be rusted only by a rust race with a corresponding gene for pathogenicity. Gene-conditioned compatibility resulting in rust susceptibility is accompanied by a common chemical component in the susceptible host and pathogenic flax rust.

The development of the capacity of an animal to protect itself by the production of antibodies comes so late in the chick that the first month of its life is a period of great vulnerability. The research of Brandly and his associates (1946) showed that the young chick has limited capacity for antibody formation till the fifth week after hatching. The embryo has little capacity to defend itself by antibody production, and accepts implants and inoculations freely. Passive immunity through transmission of antibodies from the dam through the yolk is highly variable and, if the dam lacked immunity, may be absent. Can passive immunity be built up into a more effective protection through hyperimmunizing breeder hens? Or by feeding newly hatched chicks yolk from such hens?

That the tolerance of the embryo can at least in limited measure be prolonged through its lifetime has been demonstrated through many researches, culminating in those of Medawar (1961) and Burnet, for which they were awarded the 1960 Nobel Prize in medicine and physiology. Medawar has suggested that foreign antigens entering the fetus from the mother might weaken its resistance in later life to infectious disease. Can egg-transmitted pathogens thus gain the tolerance of the developing chick and prevent chicks thus infected from developing effective antibodies against such pathogens? And thus becoming disease carriers?

Perhaps the greatest opportunity for productive research in the new laboratories will be in the evaluation of the effects of temperature, light, air movement, humidity, population density, and other environmental variables, singly and collectively on broiler development and quality in the presence and in the absence of diseases and parasites.

Of the effect of environment on disease we know little. Some diseases are seasonal. Is it because viruses can survive and are air-transmitted when humidity is high? Is it because cold (or warm) internal body surfaces permit entry to specific pathogens? It is a commonplace that many healthy humans harbor many pathogens on buccal, pharyngeal, and other surfaces; why does not infection always lead to disease? With material of known genetic composition, both host and parasite, with environments under precise control, can we determine the role of each environmental variable and of the interactions of those variables? Of temperature, light, humidity, air movement, electromagnetic forces, ionization? And of all their interaction throughout their effective range? Of stress of parasitism by one agent on the pathogenicity of another? Of nutrient deficiencies? Of antibiotics and drugs? Of age, of sex, of time and degree of exposure? We will not run out of problems, but we may reasonably hope for some useful answers.

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THE ROLE OF USDA IN FURNISHING WHOLESOME POULTRY

By Hermon I. Miller[1]

I welcome this opportunity to discuss the Agricultural Marketing Service, Poultry Division's responsibilities for providing consumers with wholesome poultry. We are happy to have our technical experts in poultry inspection participate in

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all of the discussions during this symposium, and trust that they can make a contribution to your study of research needs in the field of poultry health.

Our inspection personnel are required to review over 1-3/4 billion individual poultry carcasses a year. This opportunity gives them knowledge which should serve a useful purpose in assisting you who are interested in the research field.

In approaching the topic assigned to me I would like, first, to review the genealogy of the poultry inspection program during the quarter of a century before it became mandatory on January 1, 1959. I want to give this background because I think it contributed immeasurably in serving as groundwork for the program which began on January 1, 1959. I think we might agree that, had it not been for this background, the problems in administering the mandatory program to all of the industry would have been manyfold greater than they were.

The U.S. Department of Agriculture first began to inspect eviscerated poultry for wholesomeness in 1927. This service was requested by a soup company which needed official certification in order to export canned poultry products to Canada.

In subsequent years, a number of canning plants requested inspection service and for some time the service was limited largely to these plants. But with the development of the market for ready-to-cook poultry--which was just beginning in 1928--came increasing need and demand for inspection of poultry for sale as a fresh, ready-to-cook product.

It was during these early years, from 1928 to 1940, that USDA developed the basic regulations, procedures, and criteria for poultry inspection. The development of the inspection service was interwoven with the explosive growth of the chicken and turkey meat industries.

Inspection regulations were kept flexible enough and were modified often enough to keep pace with rapid improvements in production, in processing equipment and techniques, and in packaging and marketing. Yet they were kept stringent enough to protect the integrity of the product and promote its increasing acceptance by consumers.

By 1945, the amount of poultry being inspected for sale as a ready-to-cook product was exceeding the quantity inspected for use in canned poultry products.

During World War II, poultry inspection work expanded greatly because of the needs of the military services and an increasing consumer demand for poultry. The extension of inspection to a greater number and variety of plants during the war years made it necessary to establish printed guidelines for the expanded inspection staff and laid the groundwork for extensive changes in the regulations governing poultry inspection.

One outgrowth of the broadening of poultry inspection work during the war was the establishment of sanitary requirements for slaughtering plants. Between 1945 and 1950, poultry production took tremendous strides and hundreds of new processing plants sprang up. In these years, USDA enlarged and modernized the regulations covering sanitary requirements for slaughtering and dressing operations, and also for chilling and packaging of poultry and poultry products.

During these years, too, the consuming public and the poultry industry became more aware of the value of poultry inspection, and by 1951 eviscerating plants operating under inspection totalled 155. In addition, a large number of dressing plants were receiving sanitation inspection.

By 1955, the poultry industry had changed almost entirely to a ready-to-cook basis, and 260 eviscerating plants were operating under inspection. The USDA poultry inspection service by this time had come of age, along with the poultry industry. It was based on a long period of continuous experience with the poultry industry and with the revolutionary changes which the industry had undergone. When Congress passed the Poultry Products Inspection Act in 1957 and the administration of this law was assigned to the Poultry Division of AMS, the 30 years of poultry inspection work that had gone before and the corps of experienced veterinary inspectors--some of whom had started with the service in 1927--proved to be invaluable.

Regulations governing processing facilities and operating procedures under the new mandatory inspection program were based upon those which had been developed over the years and had proven sound in operation. Criteria for judging the wholesomeness of poultry under the new inspection law likewise was based on the older criteria developed from the laboratory findings of inspection pathologists

and tested over time. And the large corps of new inspectors needed to carry out the mandatory inspection program received thorough training from the experienced, qualified technical people who had so long worked in the voluntary poultry inspection program.

Now I would like to review some aspects of the Poultry Products Inspection Act in order to define for you the responsibilities of the U. S. Department of Agriculture under this law.

The Declaration of Policy in the Poultry Products Inspection Act says: "It is hereby declared to be the policy of Congress to provide for the inspection of poultry and poultry products by the inspection service as herein provided to prevent the movement in interstate or foreign commerce or in a designated major consuming area of poultry products which are unwholesome, adulterated, or otherwise unfit for human food."

The term "wholesome" is defined in the Act to mean poultry which is "...sound, healthful, clean, and otherwise fit for human food." The term "unwholesome" is also defined in the Act as those conditions which would be contrary to the terms defined as "wholesome."

The Act also defines "adulterated." In this connection I should like to point out that the U. S. Department of Agriculture has full responsibility for preventing adulteration of poultry under the Poultry Products Inspection Act, an authority which previously had been vested in the Food and Drug Administration under the Food, Drug, and Cosmetic Act. The definition for "adulterated" as applied to poultry and poultry products under the Act includes among other things "....If any substance has been added thereto or mixed or packed therewith so as to increase its bulk or weight, or reduce its quality or strength, or make it appear better or of greater value than it is."

In general terms, the Poultry Products Inspection Act requires USDA to:

1. Determine which poultry is fit for food. This determination must be made at the time of slaughter and evisceration. An ante-mortem inspection must be made when the Secretary of Agriculture considers it necessary, and a post-mortem inspection is required on each individual poultry carcass.
2. Promulgate regulations which establish minimum standards for premises, facilities, equipment, and poultry processing operations, and which require processors to operate official establishments in compliance with these standards. These regulations assure consumers that poultry is processed and handled in accordance with sound operating procedures and that sanitation and wholesomeness are of prime importance during processing.
3. Make certain that all poultry and poultry products are properly labeled in accordance with the law at the time the poultry leaves a processing establishment and that no misuse is made of officially identified materials.
4. Make sure that poultry is not adulterated through the addition of excess moisture, poisonous materials, or in any other manner.

In general I know that you folks here are primarily interested in disease factors--the subject we deal with in determining which poultry is fit for food. The following table is a summary of post mortem examinations of poultry in the United States for the years 1959 and 1960.

As previously indicated, inspection personnel make an ante-mortem inspection of poultry delivered to a processing plant on a lot basis. There are two principal purposes for this inspection. First, it gives the post-mortem inspectors knowledge concerning the general health of the birds they are inspecting and, second, it protects plant workers from infection from any disease that might be an occupational hazard. The law requires that each individual carcass to be given a post-mortem inspection. This takes place, of course, at the time of evisceration.

The Poultry Division has developed rather elaborate statistical information on condemnations by causes and can provide information on a plant-by-plant basis. This material is summarized biweekly. The summary of the 1959 and 1960 calendar years provided you comes from these reports. We hope that as we proceed research people will find this information of assistance in planning their undertakings.

I should point out that poultry inspectors working in a plant do not make a specific diagnosis, but rather they categorize the conditions found into broad groupings. We frequently divide condemnations into two broad groups, that is,

UNITED STATES DEPARTMENT OF AGRICULTURE
Agricultural Marketing Service
Poultry Division
Inspection Branch
Washington 25, D. C.

SUMMARY OF POST-MORTEM EXAMINATION OF POULTRY - UNITED STATES

ITEM	1959	1960
	Pounds	*Pounds*
Certified (Ready-to-Cook Weight)[1]	4,751,249,950	5,067,213,000
Condemned (NY Dressed Weight)	81,257,048	141,619,000
Percent Condemned[2]	1.41	2.28
Carcasses Condemned:	*Number*	*Number*
Tuberculosis	302,549	385,392
Leucosis	1,256,746	1,831,891
Septicaemia and Toxemia	8,818,628	11,475,815
Airsacculitis[3]	---	15,898,445
Inflammatory Processes	7,713,195	8,733,684
Tumors	305,429	328,898
Parasites	463,403	27,400
Bruises	369,373	386,190
Cadaver	1,391,973	1,346,965
Contamination	1,085,360	1,706,641
Overscald	248,231	218,895
Decomposition	44,512	73,791
Other	3,341,477	2,148,941
Total	25,340,876	44,562,948
Official Plants (eviscerating, canning, or combination)	[4] 706	[5] 854

[1] Includes rabbits.
[2] Total pounds condemned divided by 90 percent of pounds inspected less ante-mortem pounds condemned.
[3] Reported as a separate cause beginning October 1, 1960.
[4] As of January 1, 1960.
[5] As of December 30, 1960--includes 73 combination eviscerating and canning plants.

March 2, 1961

condemnations for disease conditions and condemnations due to plant operations. We consider bruises, cadaver, contamination, overscald, and decomposition as plant-caused factors. As you can see from the record before you, there is considerable opportunity for improvement in connection with the so-called plant-caused condemnations. Cadavers, for example, are largely the result of improper bleeding. Contamination is the result of careless operations. We offer whatever assistance plant management is willing to accept to reduce condemnations due to faulty plant operations.

In general terms the two most important diseases contributing to condemnations are: First, the various forms of respiratory disease, that is septicaemia and toxemia, airsacculitis, and inflammatory processes) and, second, leucosis. I would suggest that the most immediate problem is overcoming the diseases affecting the respiratory system of poultry, but that the most dangerous health problem is in the leucosis area. Our pathologists, under Dr. Graydon McKee's leadership, have an opportunity in their laboratory work to observe changing conditions in the manifestation of poultry diseases. Dr. McKee has indicated that while we have had an important problem with leucosis in connection with the broiler chicken, there is increasing evidence of leucosis appearing in turkeys.

Dr. Roy E. Willie, Chief of our Poultry Division's Inspection Branch, and his staff of veterinarians will be available to work with your symposium for its 3-day duration and can give you more specific information on this whole area of condemnations than I could hope to in this brief presentation.

In closing I would like to indicate that we are very aware of the problems the poultry industry has experienced because of the imposition of the mandatory poultry inspection program. We feel that the industry has made tremendous progress in controlling poultry diseases, but still has great need for more knowledge and more research on these problems. We have worked very closely with livestock disease eradication people, poultry diagnostic laboratory personnel, and research and extension people from land grant colleges to bring to the industry whatever assistance was available from these sources.

INDUSTRY'S PROBLEM IN PRODUCING WHOLESOME POULTRY AND POULTRY PRODUCTS

By Julius F. Bishop[1]

It is my pleasure to represent the commercial poultry industry in presenting to you some of the health problems with which we are faced. I am at a loss to understand why I was selected for this most important assignment. The only logical reason that I can give is that Georgia produces approximately twice as many broilers as any other state. Therefore, we possibly have twice as many problems as anyone else. This is very likely my only qualification for making this talk.

To try and discuss all poultry health problems is an impossibility. Health problems in 1960 cost the broiler industry alone over $65 million with $22 million losses in unsalvageable birds, and equal amounts due to medication and downgrading. Similar losses are reported in layers and turkeys with a total loss of $125 to $150 million. These losses present a serious economic impact on the poultry industry. We need help.

Because of need for answers to our problems, an effort was made to get

[1] Commercial broiler producer, Athens, Ga.

research facilities to help us solve these problems--specifically those of condemnations. The research facility emphasizing the disease aspect of the condemnation is to be located at the University of Georgia at Athens. You gentlemen are here to "brainstorm", if you please, approaches to the solution of the problem. If it is a matter of testing our breeder flocks, reliable blood tests might be set up. If it is a matter of breeding, new techniques may give the answer. If it is a matter of environmental control, the engineers can give us the answers. If it is a matter of disease control, a new or improved method of imparting immunity to birds, or a new medicant for treating diseases, or a new method of preventing its transfer might be developed. If industry can receive the basic information, we shall be in a position to apply it.

It has been said that the poultry inspection program is 5 years ahead of industry. If this is true, we are entirely dependent upon the scientific minds associated with the industry to close this gap and bring industry up to the level of the inspection program. Easy progress of industry has already been made. In the future, it will take the best scientific minds available to make any progress at all. One of the great problems is that much of the information made available by research has not been economically practical. Industry has had to comply with compulsory Government inspection. Unfortunately, not any solutions to the problems brought on by inspection have been offered from the field of research. A chronic complaint of industry is that we have paid dearly for solutions of problems by the trial-and-error method in the field, rather than by laboratory research. Also, it is said that industry is forcing progress in the poultry industry rather than being led by research.

Integration of production operations presents a problem within itself to the industry. Decisions are made on the basis of many different factors. For example: (1) The effect is considered on the overall, (2) chicks are placed because the company owns flocks, hatchery, feed mill, and processing plant, (3) by operating near capacity, overhead, and costs are kept to a minimum.

In keeping costs down in all phases of operation, many times factors that contribute to the best health of broilers are overlooked. For example, chicks are put back in the house too quickly; the house is not cleaned; the disease cycle is not broken.

With the constant pressure of competition and declining prices, industry has had to streamline and become more efficient. Thus, the industry has had to become an integrated operation which, in turn, has created additional problems. Integration makes special demands upon the poultry breeder to produce a superior hen. Hens today are outstanding in some respects but not others. We need all-around performance. We need a hen that will have good liveability and low morbidity, that produces a chick with good disease resistance, fertility, liveability of progeny, with a carcass of fine quality, and gives good yield to the processor.

The inspectors say that most of our losses are from birds exhibiting septicemia and toxemia manifestations resulting from a pleuropneumonialike organisms (PPLO) infection, chronic respiratory disease (CRD), or airsacculitis. We need PPLO-clean hens and males. We may have to go back and start from "scratch." National scale testing similar to the pullorum control program may be in order. A standard antigen and blood-testing program seem necessary.

Avian leukosis is still a real problem and is increasing. At the Georgia poultry laboratory, prior to November 1958, no visceral leukosis in young birds less than 10 weeks of age was evident. Since that time, 225 flocks involving one million birds have shown the acute form of visceral leukosis. Leukosis is also seen in laying flocks. As flocks come into production and are subjected to stress, they show signs of leukosis. Flocks from the same breeder often are affected quite differently.

Pullorum disease is still a problem. The Georgia poultry laboratory reported 29 cases in broilers during the last 4 months--only 1 case during the same period 2 years ago. There were 52 reactors in breeder flock testings during the past 4 months--only 19 reactors during the same period 2 years ago. This is particularly true when broiler chicks are in short supply, because the producer takes chances to make an extra dollar.

Flocks can be immunized by vaccination so that they will be protected against infectious bronchitis and Newcastle disease. However, in producing immunity, reaction is often so severe in heavily infected

PPLO flocks that chronic respiratory disease results. Many producers do not vaccinate, or use mild-attenuated vaccines, and run the risk of field outbreak. Results are usually calamitous.

Nephrosis, or so-called gumboro disease, is becoming more prevalent. No apparent effective treatment has been developed. Mortality is quite high in some flocks.

Debeaking of chicks costs the industry many hundreds of thousands of dollars annually. Needed is a method of control of feather picking without debeaking.

Bulk feed handling has brought tremendous problems in the treatment of sick chickens. Medication is difficult. Feed bins are usually full when sick chickens are discovered. Water medication is used and is often not effective because of carelessness in mixing. There has been no satisfactory solution to the pick-up of feed left after the birds have been sold. The delivery man can spread disease from one house to another. In using coccidiostats, we have human error in judging the amount of feed in bulk bins. We need more coccidiostats that can remain in feed throughout the growing period.

Coccidiosis is still a problem at certain seasons of the year. Needed is a standard method of determining oocyst population in litter.

Breast blisters cause severe economic losses to the industry due to down-grading of carcasses. What is the etiology? Environmental factors, genetic responsibility, size of bird, type or quality of feed consumed must be considered.

What constitutes the right amount of equipment to achieve optimum efficiency? Recent tests show startling results. The standard number of feeders is 20 to 25 per 1,000 growers. Results are better with fewer feeders. Birds have to be fed more often to compensate for the lower number of feeders.

The serviceman is one of the most important individuals connected with producing a pound of broiler meat or a dozen eggs. He is kept in a constant state of confusion and must often be a salesman, psychologist, economist, deliveryman, biochemist, nutritionist, and liar. The serviceman can spread disease from one house to another. We need a safe, simple method of sanitizing the serviceman and feed deliveryman.

One of the biggest problems confronting industry is the grower, himself. It is a constant fight to get the grower to perform up to his capabilities. Many growers remember the lush profit days and lack the efficiencies the new growers have developed. Some get into bad poultry practices and are slow to respond to the current situation. Programs of management need to be simple and practical. If the grower understands the reasons for requirements, he will do a much better job of application.

Summary

I realize that it is easy to present a problem and to expect an immediate answer. This usually is unrealistic. We all understand that research is time-consuming and at times disappointing. The poultry industry moves at such a fast pace that its members have to run to keep abreast of trends. Because of the nature of our business and the effect it has on us, I am sure that you people in the field of science and research become awfully vexed with us for our impatience. We are simply reacting normally to a business that is extremely competitive and changes each year in its approach to producing poultry economically.

We need and solicit your help in dealing with the many problems confronting us. If we are to profit by the mistakes of the past, we should give careful consideration to some of the problems of today which are not serious, but will become serious through increased poultry population through the years.

There are signs in the industry today that might alarm one concerned with the future of the industry. I feel that we have at least one thing in our favor as opposed to 10 years ago. Today, we have many more people, such as yourself, who are interested in the problems of the industry and are in a position through ability, knowledge, and research facilities, to make tremendous contributions to the present and the future of the industry. I appreciate the opportunity to present to you some of the problems confronting the poultry industry in producing wholesome poultry. I hope that I have been objective in my remarks and that through the cooperative efforts of the industry and those engaged in the field of science and research, results may be achieved that will be of a mutual benefit to all concerned.

BRIDGING THE GAP BETWEEN RESEARCH AND THE PRODUCER

By Richard Hanson[1]

Our ultimate concern is the resource--time. The tremendous opportunity for improvement of poultry health is self-evident.

What we did last year may have been as decisive as a marriage and as delightful as a honeymoon; but our life is dead if once we let last year become more important to us than this year. Nothing should be more important than the single-minded pursuit of truth. Our beliefs should be oriented in a manner such that what we find is true.

Within this rather complex situation there lies the basic problem of understanding nature in the whole. The most expedient method is to utilize many specialists with deep knowledge as a team to communicate now and frequently in the future with the large team of producers. Our poultry producers have done amazingly well in their function considering the limited amount of quantitative information they have at their command. Granted there are several who have a greater share of the necessary facts to proceed soundly in their individual operations. Now we know that this proportion in volumes of production has reached a point which indicates that the United States Department of Agriculture has an obligation and Congress has its corresponding fiscal obligation to see that many mistakes in production be avoided.

To labor this point a bit further--a review of the education practices and standards in the biological sciences should have long-term effects toward this same goal. The chief discouragement of young enthusiasm in biological sciences has been that one must master nearly the whole body of knowledge of a course of study offered or be a clever acceptor and organizer of knowledge sufficient to pass the exams. Intuition is not usually recognized; often dissent arising out of intuition is discredited. More recent acceptance of the stronger intuitions into accelerated programs at the high school level has been rewarding.

Many producers, though unsteeped in the sum total or the accepted minimum of literature, have adequate insight for their own problems. Recent advances in techniques have led many to operate outside of their domain in intuition and understanding. Without understanding why, they have lost control of their operations, especially with regard to poultry health. Entropy exists making disease or morbidity more likely without much effect from the changes in management except an increase in cost.

Research should be focused on this entropy. Through better acquaintance with this special set of conditions, one can analyze and point out the problems and their economical solutions. The study involved here is a continuum of interactions ranging from viruses to people.

Today, I would like to confine further comment to people. The most valuable tool of the USDA today is the backlog of information and continuing work in research. Without this tool it would not be feasible to recommend anything in such problems as poultry health. Eradication programs would never be successful without the wealth of information from research of the many pathologists of ARS and the State experiment stations. Fine structure and engineering work is being done by the Agricultural Engineering Research Division, regional laboratories, and State experiment stations. This work, if fully applied, would protect the US poultry industry from such heavy condemnation losses.

Let's take up the matter of communications. As we complete research projects of one form or another, many more problems become apparent that need to be solved. By this everspreading tendency in scientific work on the encircling problem, effort to communicate or share the fervor of spirit in facts restricts itself

[1] Commercial poultry breeder, Corvallis, Oreg.

or is postponed till the project is cooled by time or subsequent events and findings.

The sum total of such information is not what should be shared in communication to producers, but the experience should be. What do I mean by this? The nature of the problem is one thing; the knowledge of the problem another. Much effort is spent today sharing knowledge, especially in science. Knowledge has as many meanings as those who partake of it. There is a core of central truth, but relations of this knowledge to action varies considerably between individuals.

To share experience one takes a picture, so to speak, of a process understood and under some control. This may seem mundane, but is it? The process of any operation involves controls, many beyond our comprehension. The more controlled the process is by the human, the better he understands it if he observes what he is doing. Human control is obviously the only direct approach we have to such a process.

In our poultry industry today we need to establish sets of experience that by their nature produce better poultry and poultry products because our poultry is healthy. By seeking this goal, our birds will approach genetic capacity in proportion to the success of this overall research communication effort. There are two areas of work that have to develop before this begins. One area is the search for new facts and the synthesis of established facts into a workable set of standards and procedure. At the same time an attitude organization needs to be accomplished between researchers and producers.

Attitude organization--what is that? It might be compared to the revelation to the Apostle Paul on the Damascus road. This business of opening avenues of human electricity to permit true communication is not easy. By establishing similar motives for the researcher and the producer, simultaneously an ideal communicating situation is created.

Integrated business firms create the research to application situation directly. Large corporations gear their applied research to their immediate needs and extend with the same motive the pioneering research, though this relationship is more indirect. The success of these corporations in the development of improvements is obvious by their profits. Their research had contact with the production industry and to the consumers through the intra-corporation pipeline. Motives of research and production here are identical.

Let's open up the cover on a motive and see what makes it tick. Motives operate on the fuel of belief. People tend to establish beliefs on the scale of probabilities nearest unity of 100%, as far as they know. Not only are beliefs based on this, but they are steered by their relation to what one considers valuable. To become firmly established a belief must serve this value by one's actions as far as he knows. The application of knowledge here becomes so dependent upon value judgment and expectation that the simple truth becomes incorporated into an individual point of view.

The disadvantage we are operating under is apparent. By not having unified motives or singleness of pocketbook, our individuality (though very American and highly desirable) makes communication difficult.

What we are really talking about here is attitude. It is a big word including a before and after. Before attitude there is the situation or attitude object. The attitude in three parts is (1) affect (emotional usually), (2) knowing, and (3) action or behaviour. After this, these three aspects have their dependent variables in the same order: (1) Effect on the person, (2) perceiving responses or statement of belief, and (3) action or statement of action.

Assuming the need for attitude change or organization, we start with the second part of an attitude, action of knowing and its belief antecedent. This is the most susceptible and suitable point to consider. The idea of value is directly related to this. Advertising successes seem to be surrounded with the effort to inform the prospective customer. The information is aligned with the rest of the attitude formula in order to produce the purchase in as many cases as possible. The location of the information is such that the chances of the prospective customer to see the information are made maximum.

In producing a new or at least not commonly known fact in research, let's assume we give this fact to a producer. Mentally, the producer examines this fact in light of his present knowledge and the likelihood that this will improve his chances of reaching his goal or value. If the fact passes this test, his belief on

this subject will undergo reorganization within the intricate equilibrium of his attitude structure. During this time his mind is reorienting this belief and new information to related beliefs in a test for consistency. The changed mind results if all goes well in this process.

One problem enters the picture here. Beliefs are made with or without knowledge of true facts. These facts are in competition with previously accepted reorganized facts or wishful thinking. Without sufficient factual information human nature frustrates acceptance of true facts.

To one faced with situations that are outside the domain of his knowledge preservation of sound action concerning any problem depends upon a rapid collection of information. As this information is processed by the mind, errors might creep in unless there happens to be a similar situation with similar problems worked out nearby. Communication of facts from respected authority works well in this case if there are enough facts synthesized into a convincing form to be applied. This sort of situation exists in our colleges and universities where inexperienced students are exposed to a vast array of principles and some concrete experiences to nail them down. However, in the field of practicing production, time and energy spent in control exclude a massive communication of facts.

By packaging the communication in a package of demonstration is one possible means by which producers can be convinced of the likelihood of operational validity of facts included. The pooling of facts and their stated relationships toward optimum economy and poultry health might be considered the aims of the two southern poultry research laboratories we are herewith concerned. This should be the aim of most State experiment stations and extension services--to review and relate to industry operational improvements as soon as facts suggest or indicate them. Trade associations can help here too. The big job is the proving of the most likely improvements to the exclusion of many other possibilities.

To present the individual vital fact, its relation to procedure now practiced, should be stated and the economic alternatives be given. By this means the value relationship is given and thereby the alteration of the complicated belief patterns is more probable. When true fact fits a desire for improvement, and improvement will be made, on the basis of its importance to the value or goal of the producer. Without appeal, research results will be met with resistance or a total indifference.

Resistance to information comes by unwillingness to change a set of pat beliefs. Such new knowledge is either walled off from the inconsistent parts of an established belief pattern or ignored. Socrates had a clever way to deal with this. By having his pupils state their beliefs verbally, inconsistencies would appear--even to the one speaking--the walling off would be softened, then an adjustment of beliefs would begin or the inconsistent belief would be rejected.

Ignoring a fact might be due to the presence of a prior known contradictory fact or due to a lack of comprehension. Lack of comprehension of a fact may be a fault of the potential user, or it may be a fault of the research language or jargon. Wastebaskets collect more good work than they should. The pressure of time and the inconsistency or incongruity with values of the potential user of information prevents much communication which is of value.

In order to communicate and bring about a change in the ultimate equilibrium of poultry health and economics the producer needs to have many facts. Researchers need facts too. The two-way communication will tend to open the eyes of both ends of this program and prevent much misunderstanding. The Iowa State University approach on the poultry problems currently considered is operating with participation of producers and researchers before work begins. This is good. When the facts return from the research group the producer has a much easier time finding agreement within his beliefs than if he hadn't known what the research program was attempting.

Economic problems are foremost in the mind of most producers now. This part of his control of his operation is closely allied with the value aspect of his attitude toward poultry. Maximizing of profit or minimizing loss is the common denominator used in evaluating new information. This is the toughest nut to crack in this whole process. When information is aligned with typical economic goals, use is made of it sooner.

There are few people who operate with complete conviction. There is usually a balance or an equilibrium between positive and negative aspects of more or less

consistent beliefs. We seldom have totally consistent beliefs. In some situations we might be faced with the problem of doing something we don't believe in. We hope most poultrymen will not be faced with this unpleasantry. Added information supporting action on inconsistent beliefs of this sort tends to sicken the individual and stop the action. Justification of such an action is needed for an attitude change. Motivation creates an attitude change when it is clearly an improvement with respect to value likelihood or when resistance to change of attitude is lacking.

The strongest motive for an attitude change is free choice. Incidentally, the author and how well acquainted the reader is with his work has a lot to do with acceptance also. Two or more mutually supporting authors of one fact or group of facts makes acceptance even easier. The situation is tough enough to get research results accepted without bucking up against inconsistent belief in the potential user of the information.

The basic incentive for freedom from want is developed through a history of attitude construction that is most satisfying to those able to build on fact with the minimum of intuitive error to fill in where reliable information isn't available. The constant character of symmetry of beliefs and resistance to new facts which tip the "status quo" of belief make the information process slower. By preparing people for new information and explaining the consequences likely to occur in case the information is ignored or not used, some new facts are accepted though inconsistent with present beliefs.

SECTION II--FIELD STUDIES OF BROILER CONDEMNATION LOSSES

FIELD STUDIES OF BROILER CONDEMNATION LOSSES IN GEORGIA

By S. C. Schmittle[1]

With the advancement of the compulsory poultry inspection program that began in January 1959, it became evident that losses due to condemnation were increasing. Explanations were readily available for the pathological alterations used as the bases for condemnations. However, with the exception of a few specific disease conditions, it was practically impossible for poultry producers to determine from the inspection reports what specific diseases were responsible for the pathological alterations. With no apparent relief in sight, members of the poultry industry became alarmed at the steadily increasing rate of condemnation. Hence, in February 1960, a field study was undertaken to investigate all possible contributing factors from the breeder flocks through the growing-out and processing phases of poultry production.

Method Of Investigation

This study, which is divided into four parts, was a cooperative effort among the following individuals:

William E. Clarke, Veterinarian, Georgia Department of Agriculture, Gainesville, Ga.

John M. Kimsey, Veterinarian, ADE, ARS, USDA, Atlanta, Ga.

Dank Morris, Chief Veterinary Pathologist, Georgia Poultry Laboratory, Gainesville, Ga.

Part I. Survey of Problem Broiler Flocks

Certain contractors furnished the survey team with the names and farm locations of 53 growers in northeast Georgia who had experienced high condemnations. In all, 380,000 broilers were involved; flocks ranged in size from 2,000 to 16,500 birds. A questionnaire involving some 167 production factors dealing with all phases of the growing operation was prepared for each flock visited. Data were collected on housing facilities and management practices on all farms.

Part II. Survey of One Dealer with High Condemnation

One dealer in north Georgia was having unusually high condemnation. His average during the 4-month period from December 1959, through part of April 1960, was 8.53 percent on 1,060,300 chicks started. Many flocks had high condemnation even though management practices were good. Records for all the broilers grown from December 1959, through December 1960, were included in this survey in an attempt to

[1]Director, Poultry Disease Research Center, University of Georgia, Athens, Ga.

determine the related causes of high condemnation.

Part III. Respiratory Disease Study

At the beginning of the survey in part I, the firms agreed to submit diseased birds from flocks being studied to the laboratory for diagnosis so that the disease relationship could be established. As the study progressed it became evident that this arrangement was not dependable and in June the study was modified. It was apparent from the survey that respiratory diseases were the most significant factor associated with high condemnation. The complete questionnaire was dropped at this point in favor of a comprehensive one dealing more specifically with disease agents.

The respiratory disease study concerns 54 flocks on 43 farms with a total of 340,000 broilers. As the flocks were reported to be sick, they were visited by one of the field veterinarians. Birds were opened for examination, samples were taken for laboratory tests, and pertinent information on flock history was recorded. When the flocks were sent for processing, live specimens were brought to the laboratory for comparative tests, and the flock records completed with respect to performance and condemnation.

Part IV. Relationship Between Specific Respiratory Diseases and Condemnation of Poultry

Results of the respiratory disease study (Part III) indicated a relationship between condemnation losses of poultry and respiratory diseases. In view of this information, the records of the Georgia Poultry Laboratory at Gainesville, Ga., for the period January 1959, through June 1960, were searched for respiratory disease cases and pertinent information was recorded. A follow-up on 685 respiratory disease cases was made for flock performance and condemnation information. This information was available on 254 cases.

Results

Part I. Survey of Problem Broiler Flocks

It was anticipated that data on housing facilities and management practices, along with flock histories, would reveal factors that could be correlated with high condemnations. However, the results were not sufficiently well defined to point out specific environmental factors that did influence condemnation rate. There was considerable variation in condemnation rates among the five integrated firms. Of the five, one firm had a significantly lower condemnation rate and another had a significantly higher rate. Because one firm had a low condemnation rate, it appeared that his methods were more favorable even though they were the same as those employed by a firm with high condemnations. Records were not available for some problem flocks, which prevented the underlying causes from being determined. It could not be predicted from the data what the condemnation rates at the processing plants would be. It did not follow that a grower would necessarily have high condemnation rates in successive flocks. The most significant results showed that diseases, particularly respiratory, in the presence of adverse environmental influences, directly affected the condemnation of broilers.

Part II. Survey of One Dealer with High Condemnation

Certain breeder flocks of the 76 combinations of hatching egg sources, were identified as the parental sources of the broiler flocks with high condemnation. Condemnations were higher in broiler flocks over 7,000 in number and in flocks grown out when the average weekly low temperature dropped below 45° F. There was a definite correlation between incidence of respiratory disease and rate of condemnation. Also, a reduction in condemnation was coincident with replacement of breeder flocks.

Part III. Respiratory Disease Study

At least one specific respiratory disease was diagnosed on 35 (81.4 percent) of the 43 farms and more than 1 on 18 on the 35 farms, (51.4 percent). The incidence of respiratory diseases, alone or concomitantly, was: PPLO infection, 24; infectious bronchitis, 24; and Newcastle disease, 9. Many flocks had been vaccinated against infectious bronchitis, Newcastle disease, or both. Apparently, vaccination alone, as it was being done, was not sufficient to control the problem. After the rate of condemnation became known, a review of the incidence of air sac lesions that had been found at the time of the flock visits revealed a definite correlation with condemnations. There was no correlation between the condemnation rate and the incidence of coccidiosis lesions. There was a definite correlation between percentage of condemnation and feed conversion above 2.40.

The specific diseases causing high condemnation in one dealer's flocks were not the same as those causing high condemnation in another dealer's flocks. The incidence of respiratory diseases was peculiar to integrated poultry firms rather than determined by geographic location.

Part IV. The Relationship Between Specific Respiratory Diseases and Condemnation of Poultry

From the records of the Georgia Poultry Laboratory, Gainesville, Ga., during the period January 1959, through June 1960, 254 accessions representing approximately 1,673,400 birds were selected. They were respiratory disease cases on which condemnation rates had been obtained.

Figure I shows the monthly relationship between the incidence of respiratory

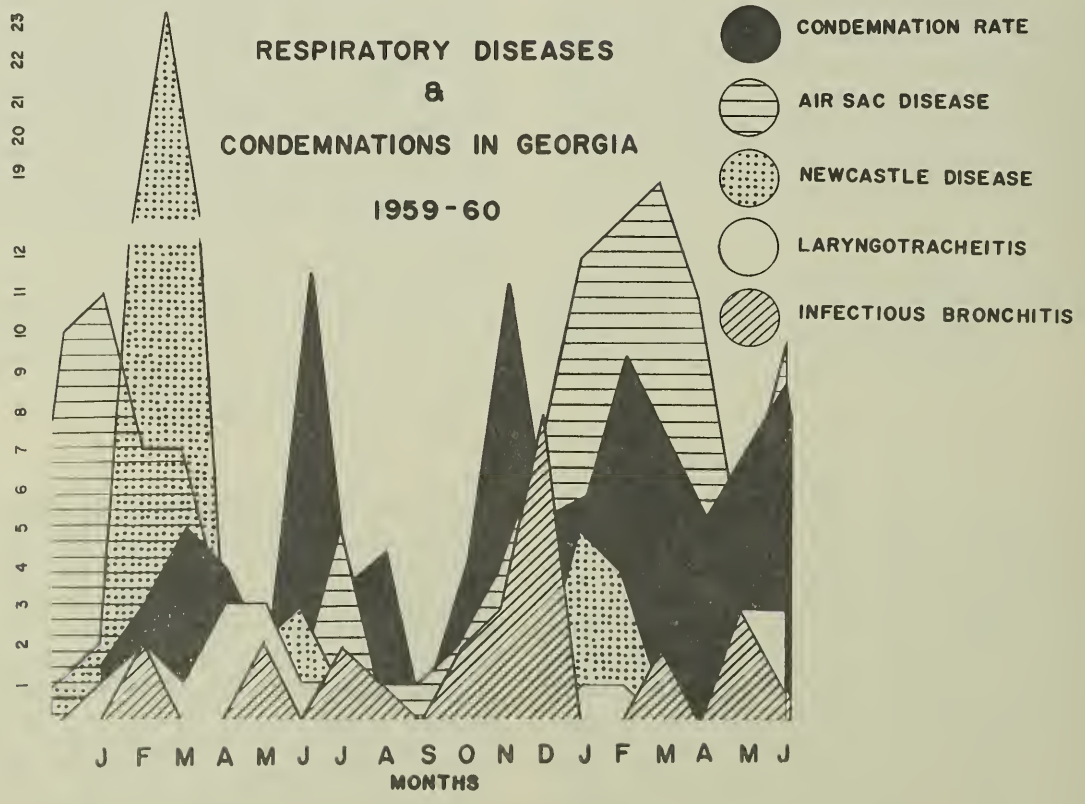

Figure 1.--The relationship between the incidence of respiratory diseases and the condemnation rate of the same poultry flocks. Compiled from the records of the Georgia Poultry Laboratory, Gainesville, Ga.

diseases and the condemnation rates on the same flocks for the $1\frac{1}{2}$-year period. In the order of incidence, the respiratory disease diagnoses were as follows:

	Cases (number)
Air sac disease	126
Newcastle disease	70
Infectious bronchitis	26
Laryngotracheitis	20

The relationship between PPLO infection and air sac disease was not determined, since the condition was well established by the time birds were submitted to the laboratory. Because of difficulties encountered in diagnosing infectious bronchitis at the stage birds are submitted to the laboratory, the incidence of this disease is not a realistic one, particularly in view of the fact that many flocks had experience with bronchitis vaccine.

As will be noted from figure 1, there is a correlation between the incidence of the various respiratory diseases and the rate of condemnation. The incidence of these diseases, as well as the rate of condemnation, reached a low level during September. This may be associated with the favorable weather conditions. Of these cases studied, it is also evident that the condemnation rates for the first 6 months of 1960 were higher than for the corresponding period in 1959, which accounts for the concern expressed by the poultry industry.

From the results obtained thus far it is felt that the following factors should be investigated to determine how they influence condemnation rates due to respiratory diseases:

1. Incidence of PPLO infection
2. Immunization procedures
3. Age at which disease occurs as it relates to market age
4. Duration of the disease outbreak

Summary

Three surveys involving 3,618,750 broilers were conducted from October 1959, through September 1960. The results show that diseases, particularly respiratory diseases, in the presence of adverse environmental influences, directly affect the condemnation rate of broilers. The respiratory diseases, in their order of importance, are: chronic respiratory disease (PPLO infection), infectious bronchitis, and Newcastle disease. With CRD, infectious bronchitis and/or Newcastle disease usually was concomitant. Vaccination procedures did not dependably control these diseases.

Respiratory diseases were correlated with condemnation rates on the same flocks. The incidence of air sac disease showed a direct relationship to less favorable weather conditions.

Conclusions

- Respiratory diseases are the most important factor related to broiler condemnations.
- Environmental (management) influences are related to high condemnations.
- The combination of adverse environmental (management and weather) conditions with the presence of disease resulted in the highest condemnations.
- Factors that lead to good records also produce good poultry and result in less condemnation.

Recommendations

- Establish PPLO-free stock.
- Initiate and maintain a disease prevention and control program specifically designed to control PPLO infection, infectious bronchitis, Newcastle disease, and laryngotracheitis.
- When a disease appears, obtain a laboratory diagnosis so that specific control measures can be taken.
- Observe good poultry management practices.
- Establish and maintain complete performance records from the breeding stock through the processing plant so that sources of problems can be pinpointed and corrective measures taken.
- Continue studies to further elucidate the basic underlying factors by which diseases contribute directly to condemantion of poultry.

A PRELIMINARY REPORT OF ACTIVITIES IN ALABAMA TOWARD REDUCING LOSSES FROM CONDEMNATION, PARTICULARLY AMONG BROILERS

By S. A. Edgar, R. N. Brewer, E. C. Mora, and J. Pruett[1]

This is a report of accomplishments to date through the cooperative effort of several agencies in Alabama to reduce losses from condemnation[2].

With the advent of U. S. mandatory poultry inspection on January 1, 1959, several new problems materialized within the poultry industry. However, during the first 9 or 10 months of inspection, the problems were no greater than might be expected from a new program of such scope.

In late November and early December 1959, reports began coming to the attention of the several State agencies that wholesome birds were being condemned and that representatives of the Inspection Service were condemning many birds which previously had been passed or cleaned and passed. Industry asked that investigation be made to determine the reasons for high condemnation, including production and processing as well as inspection and that work be directed toward a solution of the problem. Further, industry offered to support the study financially until other funds might be made available and to cooperate by assisting with such a study. In addition, the Inspection Service offered full cooperation.

A preliminary survey of several plants in Alabama in January and February 1960 revealed that (1) condemnation was alarmingly high, (2) there were irregularities in inspection, (3) there was room for improvement in processing, (4) too many diseased birds were reaching the processing plants, and (5) the greatest cause of condemnation was a generalized inflammatory process or chronic respiratory disease (CRD).

In early 1960 the Inspection Service reported that approximately 85 percent of all birds condemned at that time was for disease reasons and was the fault of producers, and that the remainder resulted from plant errors. Our preliminary observation indicated that not all condemnations were the fault of producers and processors, but that some were erroneous because of misinterpretation of existing inspection criteria and to negligence of some plant inspectors.

With this preliminary information the writers agreed to begin a condemnation study and to look critically at all segments of the industry, from the producer of the hatching egg to the final end product of the processing plants. As the study developed, work sheets were prepared and the problem was divided into five following objectives:

1. Better define wholesomeness.
2. Test accuracy and uniformity of the inspection within and between plants, using the criteria of the Inspection Service.
3. Determine farm management practices and conditions responsible for high condemnation.
4. Determine plant practices responsible for plant error and what constitutes a sanitary operation within a plant.
5. Determine what could be done to reduce losses and disseminate useful information to respective segments of the industry.

At the outset all available information regarding wholesomeness of poultry, inspection, processing and production practices, sanitation of processing, and production or grower practices that were related to high and low rates of condemnation was assembled. The literature revealed a paucity of experimental data pertaining to some of the above objectives.

Experience during 1959 had revealed that there was room for improvement in most

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[2]Personnel of the cooperating Alabama agencies involved are as follows:

Auburn Agricultural Experiment Station: Ralph Rhea, Dr. B. Bautista, and the authors of this report.

Auburn Agricultural Extension Service: James R. Hubbard, Dr. Worth Lanier, Allen J. Brown, and county agents in counties where meetings were held.

Alabama State Department of Agriculture and Industries: Dr. John Milligan, Dr. D. B. Crofts, and T. Campbell.

of our processing plants. Several years of experience with disease problems of the poultry industry in Alabama and neighboring States led the senior author to conclude that there was need of improvement in production. The following observations with respect to production were made: The winter of 1959-60 was a mild--in general, there were fewer disease problems that winter than, for example, during the winter of 1958-59; the average grower mortality was lower than in previous years; and that feed conversion improved over the previous year. There was a heavy snow on February 13, 1960, but that happened after the condemnation rate had increased.

The scope of this report covers a period from February through December of 1960.

MATERIALS AND METHODS

Inspection.--From 10 to 20 representative birds per flock were obtained at random from condemnation barrels on each visit to processing plants. In certain instances, birds condemned by each lay inspector were checked. Attached to each bird was the reason for condemnation designated by the inspector-in-charge. These birds were brought to Auburn University, Agricultural Experiment Station, and examined carefully, usually by three persons. In addition to gross observations, numerous tests were made and tissues were taken for further study.

Processing.--Processing procedures were studied, from the loading of live birds to the finished product, for each of the 17 federally inspected plants in Alabama. Also, sanitation practices, particularly with reference to the chill tanks, were investigated. Chill-tank and chill-vat water and birds were sampled at different periods and cultured for bacteria. In addition, the shelf-life of birds, from tanks and vats having different concentrations of bacteria, was determined.

Production.--A study was made of many practices and of equipment related to broiler production in several areas of the State. Information was gathered on more than 200 items related to production.

Education.--Six Servicemen's Schools were held in different areas of the State during the summer of 1960. Material presented covered condemnation causes, management and sanitation practices, equipment and housing, and others. A fact-finding conference on condemnation was held at Auburn University, December 5 and 6, 1960.

RESULTS

Inspection

The first plant visits were made on February 9, 1960, and this report covers those made through November 30, 1960. During this period, 148 visits were made to the 17 federally inspected plants. The number of visits per plant ranged from 3 to 11, and averaged 9 or 10. In addition, plants in four other States and three non-inspected plants in Alabama were visited.

Samples of 10 to 20 condemned birds per flock from 138 flocks were picked up by an Auburn University representative and brought to Auburn for study. In addition, representative samples were sent in from 73 flocks. Most of the birds sent in came from two plants. This made 2,788 birds examined from a total of 55,000 condemned birds from 211 flocks representing more than 1,250,000 processed birds. Results of findings and percentage agreement with the Inspection Service for the entire period are summarized in table 1. Average agreement with inspection on condemned carcasses ranged from a low of only 22.2 percent for one plant to a high of 91.7 percent for another (table 1). Nine samples collected or sent in from October 11 through November 30 ranged from 32.6 to 100 percent agreement (table 1). Agreement on individual flocks for the whole period ranged from 0 to 100 percent.

TABLE 1.--A study of carcasses condemned by federal inspectors in Alabama processing plants.

Plant No.	Total flocks checked	Total birds checked	Condemned	Passed	Cleaned and passed	Av. percentage agreement with Inspection Service
	Number	*Number*	*Number*	*Number*	*Number*	*Percent*
			FEBRUARY 9 TO OCTOBER 11, 1960[1]			
1	9	100	52	10	38	52.0
2	6	59	39	3	17	66.1
3	7	189	42	5	142	22.2
4	9	104	60	15	29	57.7
5	6	62	47	3	12	75.8
6	7	85	60	11	14	70.6
7	7	61	40	2	19	65.6
8	9	108	64	11	33	59.3
9	10	99	67	11	21	67.7
10	9	91	58	12	21	63.7
11	8	76	59	8	9	77.6
12	8	82	48	19	15	58.5
13	9	91	76	5	10	73.6
14	7	70	43	7	20	61.4
15	8	82	53	7	22	64.6
16	9	94	46	23	25	48.9
17	2	12	11	0	1	91.7
Sent in by industry	72	1,008	540	81	387	53.6
Total or average	202	2,473	1,405	233	835	56.8
			OCTOBER 11 TO NOVEMBER 30, 1960[2]			
1	2	20	10	2	8	50
2	1	10	8	0	2	80
3	--	--	--	--	--	--
4	1	10	10	0	0	100
5	1	10	9	0	1	90
6	1	10	7	0	3	70
7	1	10	10	0	0	100
8	1	9	3	0	0	33.3
Sent in by industry	1	[3]236	77	8	151	32.6
Total or average	9	315	134	10	171	42.5

[1]Collection Nos. 1 to 130
[2]Collection Nos. 131 to 139.
[3]236 examined out of 1,260 submitted from a flock where 53 percent were condemned by inspection.

Average agreement for all plants for specific periods is summarized in table 2. Agreement ranged from 32 percent for the period from February 9 through April 18 to 74.4 percent for September 1 through November 30. Agreement with inspection for those birds sent in by industry during the entire period was 49.6 percent. Agreement for all birds examined was 55.2 percent.

In the opinion of the authors only 243 of 2,788 birds examined should have been passed as they were, but 1,006 could have been cleaned and passed or trimmed and passed under the existing regulations. An investigation revealed that in a few instances the processor did not wish to clean and save birds or trim and save parts, but in most cases he was denied the opportunity to do so.

Most birds condemned by Inspectors were for generalized inflammatory process, septicemia-toxemia, leukosis (mostly osteopetrosis), bad fat metabolism, synovitis, contamination, and cadavers. Most of the disagreements were over birds condemned for septicemia-toxemia or a generalized inflammatory process. In some cases birds condemned for septicemia-toxemia were, in our opinion, slightly dehydrated or poorly bled, but not so poorly bled but what they would have been suitable for human consumption. Also, a good many small birds--birds slightly paler than the others--were condemned for septicemia-toxemia. Some of these were obvious variations in breeding, others were slightly stunted for some cause such as low grade coccidiosis or roundworm infections, otherwise they were normal. In no instance did we disagree on a bird that had severe coccidiosis, with anemia. There was some disagreement over birds condemned for bad fat metabolism.

Birds from the same flock, coming from split lines in the same plant, and condemned by different lay inspectors, were sampled and studied on several occasions. Agreement with them ranged from 0 to 100 percent.

With respect to this phase of the study, it was concluded that there was too much variation in inspection between plants and between inspectors within some plants. It was gratifying that, the accuracy of inspection in general improved within the period of this study and that the Inspection Service was taking steps to improve inspection accuracy. Study of groups of birds obtained since the period covered in this report, however, indicate there is still room for improvement in some instances.

TABLE 2.--Condemnation summary, by periods, 1960

Period[1]	Flocks	Total birds	Condemned	Passed	Cleaned and passed	Average agreement with Inspection Service
	Number	*Number*	*Number*	*Number*	*Number*	*Percent*
Feb. 9 - Apr. 18	27	400	128	55	216	32
Apr. 19 - July 1	52	519	353	52	114	68
July 2 - Sept. 1	29	332	222	24	86	67
Sept. 1 - Nov. 30	30	293	218	23	52	74.4
Sent in by industry	73	1,244	617	39	538	49.6
Total or average	211	2,788	1,539	243	1,006	55.2

[1]Collections are grouped in 2- to 3-month periods to show differences in percentage agreement since the study was begun.

Processor or Plant Management

In visiting the processing plants, an effort was made to determine the plant management practices responsible for high condemnation and downgrading. This study included from the catching of birds to the end of the eviscerating line. According to the Inspection Service, plant error accounted for an average of about 15 percent of all birds condemned in 1959. This was much higher than it should be and an effort was made to reduce it. Birds showing signs of contamination, overscald, misbleeding, and bruising were the most common types of plant error resulting in condemnation.

Following are some of the plant errors responsible for condemnation:

- Mishandling of birds during the catching and hanging procedures, resulting in broken bones, bruises, and dead birds.
- Misbleeds due to carelessness or lack of training on the part of "stickers."
- Overscald, because the scald water temperature was too high, or because birds were allowed to pass through the scalder twice.
- Broken bones and torn skin due to pickers.
- Contamination because of birds falling off the line in picking room and not being replaced immediately.
- Contamination because of poor initial evisceration (persons making the "buttonhole," "snip," or "pulling viscera").

Study of the many other aspects related to processing revealed that some plants lacked facilities or practices best suited to an efficient operation. Items investigated were too numerous to list here, but of greatest importance to condemnation seemed to be those associated with "sticking," "scalding," change-over (rehanging), and initial evisceration ("buttonhole," "snipping," and "drawing" operations). At stations where the above procedures occur, there was at some plants the need of closer supervision by the foreman and for well-trained substitutes who practice regularly. Initially, in some plants much of the labor received the same rate of pay regardless of job on the lines. At key points, "a bonus" or higher rate of pay for greater skill seemed beneficial in reducing plant errors. Many suggestions were made to the plant management and have been adopted.

Chill-Tank Sanitation--Water and bird samples from continuous-chill tanks and chill vats at the beginning and at the end of a half-day or full-day operation were checked for bacteria on at least two media and the shelf-life of birds studied in a limited number of tests revealed the following:

- The water source in one plant had a high bacterial count, others were relatively free of bacteria.
- There was a difference in the initial bacterial counts in tank water between certain plants, which reflects a difference in the thoroughness with which tanks were cleaned and disinfected between daily operations.
- Bacterial counts of continuous chill-tank water usually increased to some extent during a day's run. This was not necessarily the case for bacterial counts on birds coming from those tanks.
- Bacterial counts of water were higher in the first tank of continuous chillers than in the third tank.
- Birds passed through the chill-tank water toward the end of a day's run had no more bacteria on them than those passed at the beginning of a day's operation.
- There was a marked decrease in the prevalence of certain species of bacteria on birds after they had passed through the continuous chiller.
- There was no indication that an increase in the bacterial count in chill tanks during a day's run with less than a gallon of water per bird influenced the shelf-life of carcasses. Improvement in length of shelf-life was not associated with an increase in the amount of water up to 1 gallon per bird.
- The bacteria in chill tanks and continuous chillers or on birds immediately after processing were not the same as those predominantly associated with decomposition.

Production

Fifteen growing operations in Alabama were selected for investigation after the records of a larger number had been studied. The group selected included growers having consistently high condemnations and some having low condemnations. The management practices

of those growers were investigated to detect those practices related to high condemnation, and the study involved more than 100 flocks during the period of this report.

Although there is a great deal of information yet to be obtained from the data collected, the following factors appear to be related to high rate of condemnation:

- Large flocks, wide and long houses.
- New, inexperienced growers.
- Overcrowding of chicks under brooders.
- Disease outbreaks.
- Persistance of disease symptoms until the time of slaughter and lack of prompt diagnosis and proper medication.
- Moderate to severe infections of roundworms.
- Moderate coccidial infections.
- Reduced feedings of chickens, only 1 or 2 times per day.
- Poor feed conversion.

Factors which seemed to have no bearing on condemnation rate were:

- Distance between houses.
- Distance between poultry farms.
- The type and size of brooder.
- Use of brooder guards and age of birds when guards were removed.
- Spacing of brooders.
- Time length of preheating brooders, provided it was 8 hours or more.
- Routine worming program.
- Supplemental heat.
- Debeaking.
- Use of a coccidiostat.
- History of a previous disease outbreak on the farm.

In summary, it was found that there was room for improvement in inspection as well as in processing and production. For the early part of the study, approximately 50 percent of the problem was the fault of the Inspection Service.

Respiratory infection resulting in generalized inflammatory process of air sacs was the most common disease cause for condemnation.

There was almost a universal need for better relations between plant management and the Inspection Service, and for better liaison within the Service. Each party failed to communicate regularly over points of disagreement or problems where explanations were needed. There was fear of retaliation. During the past 9 months improvements have been made in each of the areas.

Certain aspects of this study have been reported in greater detail in the Alabama report at the "Fact Finding Conference" held at Auburn University in December 1960; in reports to the Alabama Poultry Improvement Association, who have given financial support to the study; and in reports to the Agricultural Marketing Service. This study is still in progress.

SURVEY OF POULTRY CONDEMNATION

By M. S. Cover[1]

On January 1,1959, the Poultry Inspection Service, a division of the Agricultural Marketing Service, began to carry out the requirements of the "Mandatory Poultry Inspection Act." This law requires the Inspection Service to certify as wholesome all poultry and poultry products for retail trade that are shipped across State boundaries. It was inevitable that such an undertaking would present a number of problems. However, few if any people predicted the impact that it would have on the poultry industry. Perhaps no other event in the history of the industry has produced such a flood of controversy.

The mandatory inspection of all poultry has awakened the industry to errors and omissions in poultry husbandry. The 2 to

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3 percent loss due to condemnation is a new factor to many operators and now becomes a part of production costs. This new factor in production cost must be reduced.

The industry, under the stimulus of the University poultry specialists and the Extension Service, has started programs to reduce disease incidence by improving sanitation practices on the farm. Growers are more careful about: (1) vaccination techniques, so that proper immunity is produced against current diseases, (2) dissemination of disease by farm-to-farm visitation, (3) proper care of birds in all periods of stress, and (4) use of more strict sanitation between flocks and in day-to-day management.

In order to ascertain to what extent factors of management and disease control influence the rate of condemnation, the University of Maryland and the University of Delaware were asked to investigate these factors. A survey was completed on September 15, 1960. It showed the following:

1. Large numbers of chicks around one stove tended to increase the rate of condemnation.
2. Condemnations were less in narrow houses (4.39 percent) than in wide houses (6.98 percent).
3. Condemnation in houses heated by individual gas stoves was higher than in houses heated by oil stoves.
4. Average rate of condemnation was 3.32 percent in houses having solid partitions between pens and 7.87 percent in houses having open partitions.
5. The use of a definite sanitation plan, such as litter removal between flocks and house disinfection, showed a marked reduction in condemnation rate. In washed and disinfected houses, 86 percent of the flocks had less than 4 percent condemnation. In non-disinfected houses, 63 percent of the flocks had less than 4 percent condemnation.
6. The average condemnation rate was 2.39 percent in flocks receiving the recommended vaccination program and varied from 3.56 percent to 9.23 percent in flocks receiving other vaccination programs.

Surveys of this type do not produce conclusive evidence. However, certain trends within the data indicate that good management and disease control do reduce the rate of condemnation. A second survey of 500 flocks is almost complete and indicates the same trends. Many people in the industry have followed a more vigorous sanitation and management program with considerable success.

BROILER CONDEMNATION STUDIES IN LOUISIANA

By J. M. Dixon[1]

Because of the heavy and increasing condemnation of broilers in Louisiana in 1959 and in early 1960, the broiler industry of Louisiana asked the Louisiana State University for assistance in reducing the condemnation rate. In March 1960 a survey was started in the Hammond and Alexandria broiler areas to determine the causes of broiler condemnations. From this survey it was determined that the broiler grower, inspector variation, the processing plant, and the breeder hen were the groups responsible for most of the condemnations.

In September 1960 the following program was started with most of the broiler producers and all of the inspected processing plants in Louisiana participating:

1. ENVIRONMENT--Weekly inspections on the farm of the husbandry

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practices by the pullorum control group of the Louisiana Livestock Sanitary Board (LLSB) and the Louisiana State University (LSU) Poultry Industry.

2. DIAGNOSIS--Biweekly Inspections on the farm for diseases in the flock by veterinarians from the LLSB--Diagnostic Laboratories.
3. PATHOLOGY--Gross and histopathology of passed and condemned carcasses by LSU--Veterinary Science and by LLSB--Diagnostic Laboratories.
4. MICROBIOLOGY--Culture for disease agents in condemned and passed carcasses by the Northwestern State College--Department of Microbiology, at Natchitoches, La.

To March 1, 1961, the condemnation rate (head count) has decreased and according to the outlook, will continue to decrease. The condemnations on the current program were as follows:

Postmortem:	Percent
Grower	56
Processing plant	19
Breeder hen	17.5
Hatchery	4
Catchers and haulers	2
Inspectors	1.5

Antemortem:	Percent
Hauler	86
Processing plant	9
Breeder hen	5

Of 28 flocks processed under this study, condemnations were as follows (head count):

Flocks	Condemnation
Number	Percent
17	Less than 1.0
9	1.0 to 1.99
2	2.0 and more

Condemnations have been reduced, because the broiler grower is doing a better job and the inspection service has been stabilized. But, to whom should carcasses condemned for leukosis be charged?

SECTION III--DISEASE CONSIDERATIONS

AIR SACS--THEIR DISTRIBUTION AND MICROSCOPIC STRUCTURE

Alfred M. Lucas and Effie M. Denington[1]

I am happy to review with you our recent studies on the anatomy and histology of the fowl, especially in respect to the respiratory system. It is true now, as it was 50 years ago, that we need to understand how the body is assembled in order to grasp fully the significance of what we learn in physiology, pathology, bacteriology, virology, and even poultry management. Dependence upon what has been written for man and mammals can lead to serious errors when applied to the fowl; for example, there is nothing in any mammal equivalent to the air sacs of birds. We are forced to stock pile our own basic information on the fowl and when we do this, we will receive the dividends in future years as we face and meet new disease problems in poultry.

The lung of mammals is composed of a system of branching tubes that terminate in thousands of blind sacs, called alveoli. The air is pumped in and out. However, mammals never breathe complete fresh air because, before it reaches these terminal sacs, it is many times diluted with depleted air. The birds, as they arose from the reptiles, never adopted the mammalian model--it was too inefficient for these active creatures of the air. Instead, the birds evolved an anatomy that could bring fresh air adjacent to the circulating blood. Basically, the avian lung is open at both ends; one end connects with the trachea and the other end is represented by a ring of openings leading to the air sacs. The air arriving by way of the trachea is shunted through the lungs to the air sacs and then with expiration filters back toward the trachea by way of numerous small tubular air canals, called parabronchi. It has been said that under vigorous breathing the bird can have a continuous stream of air going in one direction through the parabronchi (Hazelhoff, 1951).[2] If true, this would bring to the circulating blood, air with a higher oxygen content than in mammals.

Many theories have been suggested concerning the functions of air sacs, some reasonable, some fanciful. Mueller (1908) summarized many of these theories and a few others have been added since his publication.

Air sacs:

1. Function to reduce the specific gravity of the avian body,
2. Act coordinately with the lungs in breathing movements,
3. Serve as a bellows for the lungs,
4. Aid in controlling body temperature,
5. In conjunction with the lungs evaporate water from the body,

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[2] Names and years in parentheses refer to literature Cited at end of this paper.

6. Supplement and extend the respiratory function of the lungs,

7. Serve as a manometric organ like the swim bladder of the fish,

8. Store air for vigorous flight,

9. Act as a resonator organ to increase volume of sounds made by the bird,

10. Act to rigidly fix the wings in an extended position when gliding,

11. Erect the feathers,

12. Prevent shallow diving birds from going too deeply,

13. Aid the intestines in mixing food by setting up alternating positive and negative pressures,

14. Cool the testes to assure production of functional sperm,

15. Dampen the vigorous inhalation and exhalation movements of air through the lungs,

16. Can shift the center of gravity forward during flight by contracting those in the anterior parts of the body and expanding those in the posterior parts, and

17. Act antagonistically; the thoracics expand on inspiration and at the same time the cervical, interclavicular, and abdominals contract.

Among the 17 items listed, the first two may be accepted as axiomatic; their reality can be confirmed by measurements and direct observations; others, such as 10 and 11, can be laid aside as being improbable.

The air sacs of the chicken are typical for those of birds in general and will be described first. The recent report by Akester (1960) on chicken, duck, and pigeon agrees with our own observations. Cover (1953) and Rigdon, et al. (1958) have described the air sacs in the turkey. King (1956, 1957) studied the chicken and has been interested in the relationship of the anatomy of the respiratory system to its physiology (Biggs and King, 1957).

A pair of cervical air sacs arise from the anterior margins of the lungs and are located above the esophagus. There is a fusion of the right and left components, so that one may consider them to be a single sac rather than a pair. Numerous outpocketing or diverticula come from each cervical sac. These penetrate the bones of the first two thoracic vertebrae as well as the attached ribs. Four longitudinal trunks extend parallel to the neck vertebrae and cross-connections lie both between and within the bones of the neck. The first two vertebrae of the neck (axis and atlas) are not pneumatized according to King (1957). In addition, there are extensions upward into the neck muscles.

The interclavicular air sac lies ventral to the esophagus, fills the spaces above and below the trachea, and has numerous diverticula to the anterior part of the body. This sac, although single, arises by a complete fusion of four embryonic sacs which become reorganized into a medial and two lateral chambers (Locy and Larsell, 1916). Diverticula fill spaces above the heart and around the major vessels, between the heart and sternum and above the proventiculus. In part, the keel and lateral plates of the sternum are pneumatized as well as the manubrial spine; also the clavicle and coracoid bones. A narrow canal leads from the thoracic cavity into the muscles around the shoulder joint, where the sac expands to form several axillary diverticula. From one of these a further extension penetrates the humerus and extends throughout its length, but none of the bones beyond the elbow are pneumatized. The axillary diverticula lie between two large breast muscles, the pectoral and supracoracoid. These diverticula are economically important because (1) if the chicken reflexly moves its wings while passing through the scald water, it draws this water into a region of tissue used for food and (2) if an infection is present, only an extremely thin membrane separates the purulent content of the sac from major arteries, veins, nerves, tendons, muscles, and joint capsule.

The anterior thoracic air sac lies ventral to the lungs. Medially it is covered by the

transverse septum that separates the hepatic cavity from the thoracic cavity and laterally it is closely adherent to the thoracic wall. This air sac has no diverticula.

Two air sacs have their origin from the posterior margin of the lung--the posterior thoracic air sac and the abdominal air sac.

The posterior thoracic air sac, the smallest of all sacs with no diverticula, lies behind the anterior thoracic air sac and ventral to the caudal end of the lung. Its medial wall presses against the abdominal air sac.

The abdominal air sac, the largest of all sacs with numerous diverticular, thoroughly encloses all of the abdominal visceral organs except the liver, which lies in a closed body cavity of its own. The gizzard, intestine, caeca, rectum, spleen, mesenteries, and testes are pressed between the medial walls of the right and left sacs. The distal end of the duodenum and pancreas are encircled in reflected folds of the sac. All diverticula spring from the dorsal margin; inguinal diverticula surround the hip joint and extend for a short distance on the inner surface of the leg bone. The femur is not pneumatized in the chicken, but it is in the pheasant, a close relative of the chicken. A large diverticulum lies above each kidney and extensions from these penetrate the lumbar and sacral vertebrae and part of the ilium.

Other species of domesticated birds show some differences from the chicken. The turkey, unlike the chicken, has a pair of cervical air sacs. In the thoracic region there is only one pair of sacs: Cover (1953) and Rigdon, et al. (1958) considered that the one remaining pair represents the posterior thoracic; whereas, Lucas, et al. (1959) considered it to be the anterior thoracic. A study of the embryology of the turkey is needed to settle the point.

The White Pekin duck has relatively larger lungs than those in chickens, and its interclavicular air sac is more extensive than that in the turkey. This air sac is accompanied by a diminution of the anterior and posterior thoracic air sacs. The cervical sac is paired (Rigdon, 1959 a).

Latex injections of an adult pigeon demonstrated that the keel was entirely pneumatized. This is not true for the turkey or chicken. In the pigeon the lateral plates of the sternum and almost the entire ilium are pneumatized.

Birds have increased the efficiency of their respiratory system by the elaborate system of channels and sacs just described, but like any complicated mechanism, the system can get out of repair rather easily.

Disease and the pathology that accompanies disease is molded by the histology of the tissues and the reactivity of its cells. The available descriptions in the literature of the normal histology of the sac wall have been brief indeed.

The wall is composed of three tissue layers, an endodermal epithelium, continuous with the lining of the lung, a mesothelium continuous with the visceral peritoneum of the body cavity, and a cushion of connective tissues between the two epithelial layers. The exceeding thinness of the air sac wall makes it possible to study this membrane both as a stained transparent whole mount and, of course, by means of the usual cut and stained sections.

Probably the most conspicuous feature seen in the whole mount is the presence of large areas on the ventral side of the sac that have no blood supply. The vessels enter the upper half of the abdominal air sac by way of its dorso-medial and lateral attachments. These vessels are local in origin from the points of attachment to the lateral body wall and are not derived from vessels within the lungs. The capillaries are spaced far apart and many cells between the vessels are without adjacent or nearby blood supply. An elaborate anastomosing capillary network, similar to that seen in the mesentary, is absent in the air sac. At the periphery of the vascular area are many small vessels that end blindly or terminate as loops. The blood, therefore, is returned sometimes to the same vessel from whence it came.

There is no significant difference in the histology of the vascularized and avascularized areas of the sac wall, except in some few details. We would like to pass on to the physiologist the question, "How do the cells in the avascular area survive?" It is conceivable that the oxygen requirements could be satisfied by the inspired air, but what serves as the transport system for food supply and for the elimination of waste products? It can be observed that yeast cells survive equally well whether they fall in a vascular or avascular area.

Let us examine first the histology of the avascular part of the air sac. The wall is about 6 microns thick and includes three layers. The inner epithelial lining of the sac is composed of flattened hexagonal shaped cells, each with a flattened oval-shaped nucleus. These nuclei are for more

abundant than those for any of the other cells of the membrane and to anticipate what will be emphasized later, it is these epithelial cells that appear to be the chief reactive tissue to foreign body stimulation.

The outer epithelial covering is composed of large flattened cells, a micron or less in thickness. The diameter of these flattened cells is two to four times greater than that of the epithelium on the opposite side of the membrane. The nuclei take the stain intensely and are often elongated and kidney-shaped. Looking through the thickness of the membrane, it is readily possible to separate the nuclei from each of the epithelial layers.

Nearly all of the space between the epithelial layers is filled with two types of connective tissue, collagenic and elastic. The individual collagenic fibers are grouped into bundles that spread out in sheets or form an intertwining network. The bundles appear straight or wavy depending on how much they are stretched. The chief function is to provide tensile strength for the thin sac wall. The elastic fibers are single strands of uniform diameter. They are much fewer in number than the collagenic bundles and in surface view form an irregular network; in sections it was found that many elastic fibers stretched transversely across the membrane between the epithelial surfaces. Elastic fibers are abundantly present in the lungs and air sacs and have the function of collapsing these structures when the air pressure is reduced.

When blood vessels are present, they lie entirely within the connective tissue layer. To accommodate the vessel, one or both epithelial surfaces may be elevated. Around and adjacent to the vessels there is an increase in both collagenic and elastic tissues. Circular and longitudinal smooth muscles are conspicuous in the walls of the larger arteries. The endothelial lining is composed of flattened cells that have their long axes parallel to the axes of the vessels. The long dimension of the cell is usually several times greater than that of the width.

Bundles of nerve fibers are distributed among the connective tissues of the air sac wall but only in those areas where blood vessels are present. Thus far, they have never been found in the avascular parts of the sac. Are they motor or sensory, somatic or autonomic? The meager facts that we have will be reviewed briefly. A bundle of nerve fibers may run parallel to a group of vessels, but in many instances the nerve may cut across major vessels. The same is true of individual fibers that may extend for a considerable distance crossing both large and small vessels and then disappear in the neighborhood of some vessel. The exact nature of the termination has not been seen, but it is clear that there is no arborization or laminated end-bulb. Probably there is merely a simple termination of the type found on the smooth muscles that move the feathers. The fiber axons are small and those within a bundle are of equal diameter.

The anatomic evidence suggests that these are autonomic motor nerves. Intrinsic ganglia cells have been seen adjacent to blood vessels. Some fibers terminated there (preganglionic fibers); other fibers arose from these cells and followed the course of blood vessels (postganglionic fibers), and terminated near vessels. What has been seen thus far is suggestive of the parasympathetic component of the autonomic nervous system. If true, then the nerves are probably vasomotor regulators of the air sac blood supply, but other experiments would be required to determine if they have a constrictor or a dilator function. In view of the fact that intrinsic ganglion cells are rarely seen, it is probable that some, perhaps many, of the fibers belong to the sympathetic component of the autonomic system. Then the question arises, "Why is the organism equipped with vasomotor regulators when approximately half of the abdominal air sac is without blood vessels of any kind or size?"

A diligent search was made for a third type of connective tissue; namely, reticular fibers. The cells associated with these fibers are important in body defense and constitute one facet of the reticuloendothelial system. It is important to know if the air sacs are equipped with this defense tissue. Another manifestation of the reticulo-endothelial system is the macrophage. In our preliminary studies we have had negative results in our efforts to reveal the existence of a reticulo-endothelial defense system. The same techniques that clearly revealed the reticular fibers around the liver sinusoids failed to show them in the abdominal air sac. Trypan blue injections intensely colored most tissues of the body but revealed no macrophages in the air sacs. In the normal healthy air sac, heterophils characteristic of inflammatory reactions have not been found in the abdominal air sac, but they will appear in great numbers under stimulation.

Yet we do see evidence of reactivity in the air sacs and we see opaque areas or milk spots. When areas like these are observed in the peritoneal tissues of mammals, accumulations of macrophages are usually the cell type present in greatest abundance (Mixter, 1941). These lie in the subepithelial connective tissues, but in the air sac from what we have observed in initial and minimal reactions to stimuli, the reactive tissue is the endodermal epithelium.

We have already described the typical flattened epithelial cell. Under stimulation by agents outside the body, these cells divide and as they become crowded they change to a cuboidal and later to a columnar shape. These epithelial plaques sometimes involve only a few cells; other times they involve large areas with thousands of cells. They occur in both avascular and vascular regions of the air sac, but with probably some predisposition for the vascular area. Sometimes the columnar cells are ciliated, and mucous (goblet) cells appear interspersed among the ciliated cells. In other instances the plaque is composed of a stratified layer of cells, in which the surface cells are spherical. On a few occasions a foreign body or organism could be seen in the center of the plaque, but in most instances there was no visible evidence of the stimulating agent. The plaques are usually round or confluent masses, but may in some cases stretch as a thin line an inch or more across the avascular inner surface of the air sac. In appearance it resembled a streak made across a culture plate.

In the search for reactive cells it was possible to find among the connective tissue fibers a few scattered cells with the shape of histiocytes and many larger cells resembling fibrocytes. It was possible to find tissue mast cells in both the avascular and vascular areas. In the latter region they usually aligned themselves parallel to the outer vessel wall. Perhaps under severe stimulation these various types of connective tissue cells could be aggregated and rendered reactive. But the mild type of reaction we have observed thus far has usually involved only the epithelium, although occasionally typical lymphoid foci may be seen associated with blood vessels. In respect to their significance for the avian body, see Lucas, 1949; Lucas, Craig, and Oakberg, 1949; Lucas and Oakberg, 1950; Oakberg, 1950; Payne and Breneman, 1952; Biggs, 1957; and Denington and Lucas, 1960.

The ciliated cells in the trachea of rabbits were able to phagocytize foreign particles (Ropes, 1930); a similar reactivity occurs in the air sac epithelium in birds. A chicken was placed in a large plastic bag and allowed to breathe powdered animal charcoal for 1-1/2 hours. The specimen was opened immediately thereafter. In the trachea there were strings of carbon laden mucus. Rigdon (1959 b), after the injection of india ink, found carbon particles between the cells and a few particles of saccharated iron oxide inside the epithelial cells of the duck trachea an hour after injection.

In our material carbon particles were absent from the air capillaries and from the lumen of the parabronchi but were present in the abdominal air sac. Even during this relatively short period of time a large number of particles had been phagocytized by the nonciliated cells. These cells were the flattened type characteristic of a normal epithelium. During this 1-1/2 hours of treatment, an inflammatory reaction had developed. Heterophils were abundant both in the epithelial and subepithelial layers. The heterophils in their course of migration from the blood vessels to the free surface of the epithelium ingulfed particles of carbon. The "pus" cells were deposited within the cavity of the air sac.

In areas where patches of ciliated epithelium with goblet cells were present, the carbon was trapped by the mucus and moved along by the cilia; but it is doubtful if the ciliated patches in a normal chicken are extensive enough and have sufficient continuity to actually transport particles of this sort to the mesobronchus of the lung. Most of this journey is uphill.

Examination of the abdominal air sac membrane indicates that, under normal conditions of health, the endodermal cells are not ciliated. However, under the action of mild irritants, mucous and ciliated cells may be brought into existence through a process of cell division and differentiation. Our preliminary examinations thus far would indicate that the extent of ciliation varies among the different air sacs but that ciliation is generally absent from pneumatized bones. A study of pathogenesis of air sac infection is certainly needed, as well as a study of recovery processes. For anyone who plans an investigation of the ciliated epithelia of the respiratory tract, the following reviews and technics may be of some value: Lucas, 1932, 1933 a, b; Lucas and Douglas 1934, 1935; Lucas and Lucas, 1952.

In closing I would like to offer one admonition, and that is to approach the disease problems that lie before you on the normal histology, reactive tissue mechanisms and pathology of poultry primarily from a study of the fowl itself and not biased or applied second-hand from what others have told us about such tissues and mechanisms in mammals. It would seem to me that we could even afford to forget a great deal of didactic and dogmatic training of our past and let the fowl itself give us the answers to its own problems based on its own anatomy, histology, and tissue reactivity.

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ROLE OF THE PRINCIPAL ACUTE VIRAL RESPIRATORY INFECTIONS IN THE CRD-AIR SAC SYNDROME

L. C. GRUMBLES[1]

Even before chronic respiratory or air sac disease were known, there was a belief that some unknown chronic respiratory condition was associated with infectious bronchitis. In fact, chronic respiratory disease (CRD) was first described by Delaplane (5)[2]

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[2] Figures in parentheses refer to References at end of this article.

as a result of his efforts to clarify a "chronic" form of infectious bronchitis following immunization procedures. His efforts to isolate infectious bronchitis virus (IBV) from birds with lingering symptoms following intentional exposure to infectious bronchitis resulted in the isolation of another agent, later recognized as a pleuropneumonialike organism (PPLO). Thus, from the very beginning infectious bronchitis and chronic respiratory disease have been closely associated.

At a conference in 1952 sponsored by the United States Department of Agriculture (4), there was much discussion about the effects of Newcastle disease vaccination on air sac. Industry representatives emphasized the occurrence of air sac following vaccination for Newcastle disease and laryngotracheitis. At this conference little or no experimental data or valid observations were presented on this important question. Since 1952, there has been a slow, but continuous, accumulation of research data and critical observations on the relationship of acute respiratory infections and the CRD-air sac syndrome.

The purpose of this report is to present briefly the known information on the role of Newcastle disease, infectious bronchitis, and laryngotracheitis on the chronic respiratory disease-air sac problem. In the preparation of this report an effort was made to review most of the available published scientific literature on the subject. However, no effort was made to cover articles in popular or trade journals or magazines. Also, random incidental observations buried in scientific articles whose titles give no clue that information on this subject is included may have been missed, because to review all articles on CRD in detail was prohibitive.

Surprisingly few experiments have been intentionally designed to show the relationship of acute viral infections to CRD, but many field cases have been studied and many individuals have made critical observations.

CONTROLLED EXPERIMENTAL RESULTS

In 1952 Grumbles and coworkers (10) observed an accidental infection with Newcastle disease in experimental birds that resulted in a chronic respiratory disease. Then intentional exposure to infectious bronchitis was used as a method of making PPLO infection manifest itself in studying transmission of infectious sinusitis in turkeys to chickens. This work, in light of our present knowledge about almost universal infection of commercial chicken flocks with PPLO, may not have answered the main question under study, but it did provide well controlled conditions for determining the effects of bronchitis virus on birds already infected with PPLO. From the results of these experiments it was obvious that infectious bronchitis virus "triggered" CRD.

Adler (1) working at the University of California, reported in 1955 that intentional exposure of PPLO infected chickens to infectious bronchitis virus resulted in a more severe disease, as manifested by more severe symptoms and lesions. However, he reported that the birds developed no pericarditis or perihepatitis.

In 1958 Gross (9) reported on the role of Escherichia coli in the cause of chronic respiratory disease. In this study he used combinations of PPLO, "Crawley virus," infectious bronchitis virus, and various strains of E. coli. He used all combinations of the agents at various time intervals of exposure. Gross concluded that all combinations of respiratory disease agents increased the pericarditis producing ability of E. coli. He showed that infectious bronchitis virus especially had this ability and that IBV infected chickens were more susceptible on the eleventh and twenty-second days after inoculation. He reported that "Crawley virus" infected chickens were most susceptible to E. coli 2 to 4 days after inoculation with the virus.

Adler and coworkers (2) in 1960 reported that, "Latent egg-born M. gallisepticum was activated by infectious bronchitis virus under natural and experimental conditions." This statement was included in a general review article and the authors did not give the data on which the conclusion was based. In this same article they also stated, "Vaccination with infectious laryngotracheitis

sometimes produced severe hemorrhagic tracheitis in birds with Mycoplasmal infection of the trachea." Again, the details or the data on which this conclusion was based were not given.

The references cited above were the only ones found in the literature reporting controlled experimental results on the relationship of the acute viral infections to CRD. However, there have been a number of reports of laboratory studies on naturally occurring field cases of the CRD-air sac syndrome.

RESULTS OF LABORATORY STUDIES ON FIELD CASES

In 1953 Fahey and coworkers (8) in Canada reported their observations on field cases during a 2-year period. Of 87 field outbreaks of CRD, 40 flocks had been vaccinated for Newcastle disease and 47 had not been vaccinated. From these results the authors concluded that vaccination played no part in causing CRD symptoms. These authors observed an overlapping of "vaccination takes" and the beginning of CRD symptoms, but concluded that this was a coincidence because vaccination was often done at about the age (5 to 8 weeks) when CRD symptoms would start anyway. They concluded, "The present observations refuted the contention that vaccination stress per se is a stimulatory factor in the pathogenesis of chronic respiratory disease."

In 1954 Wasserman and coworkers (14) attempted virus isolation on field cases of air sac disease submitted to the Diagnostic Laboratory at the University of Rhode Island. In this study infectious bronchitis virus was isolated from 9 of the 20 cases studied and Newcastle disease virus was isolated from one. These workers assumed that infectious bronchitis or Newcastle disease virus may have caused the so-called air sac lesions. They concluded that the gross lesions commonly ascribed to air sac may co-exist with Newcastle disease or infectious bronchitis. This study did not recognize the "triggering" ability of the viral agents. In 1955 Fahey (5) reported his studies on 187 cases of CRD with air sac complications. He found infectious bronchitis associated with 37 cases and Newcastle with 11, but found no case infected with laryngotracheitis.

In 1957 Van Roekel and coworkers (13) in Massachusetts did a comprehensive study on a number of field cases. They selected cases where respiratory symptoms had been present longer than 2 weeks and placed emphasis in their selection on so-called air sac cases. They attempted to exclude uncomplicated infectious bronchitis or Newcastle disease. This report included the laboratory findings on 77 consignments. Newcastle disease virus was identified in 52. Serological procedures indicated that most flocks whether vaccinated or not had an adequate immunity against infectious bronchitis. These authors concluded that both Newcastle disease and infectious bronchitis play a significant role in the occurrence and severity of CRD.

Sullivan and coworkers (12) published a report in 1957 of laboratory studies on naturally occurring outbreaks of CRD on six farms. They found PPLO in birds from nine flocks. In all flocks evidence was also found of passed or concurrent bacterial or viral infections. These authors concluded that their results indicated the ability of viral infections (ND and IBV) as well as vaccination procedures (ND and IBV) to incite or influence the course of CRD in the field.

PERSONAL OPINIONS AND MISCELLANEOUS OBSERVATIONS

Jungherr and Luginbuhl (11) in 1952 remarked that vaccination against Newcastle disease is often blamed for air sac infection. They further pointed out that mixed infections are common in field outbreaks and stated, "...the existence of air sac infection as an independent entity is problematic."

There is almost unanimous agreement among research workers on the CRD-air

sac syndrome that Newcastle disease and infectious bronchitis virus play an important role in this disease complex. In many places, statements to this effect are made by various authors as accepted general information. Whether or not other virus infections have a similar function has not been too well established. As previously indicated, one author (Adler 1) has stated that laryngotracheitis vaccination may be more severe in birds infected with Mycoplasma when compared to similar birds that are not infected with these organisms. Beasley and coworkers (3), working with PPLO infections in turkeys, reported that a combined infection with the ornithosis virus and PPLO resulted in more extensive and severe lesions than either agent alone.

In 1954 Fahey and Crawley (7) reported the isolation of a virus which they concluded, "... is apparently involved in the etiology of chronic respiratory disease..." These results have not been confirmed by other investigators with the exception that Gross reported that the "Crawley virus" enhanced the ability of E. coli to produce pericarditis. At this stage of our knowledge no definite association between this virus and the occurrence of CRD in the field has been established.

CONCLUSIONS

Chronic respiratory disease was first recognized by efforts to clarify persistent respiratory symptoms following infectious bronchitis immunization. Thus, from the first, CRD has been closely associated with acute viral respiratory infections. Surprisingly few experiments have been designed to show the role of infectious bronchitis, Newcastle disease, or laryngotracheitis in CRD-air sac infections. However, controlled experiments, careful laboratory studies on field cases, and critical observations have conclusively revealed that acute viral respiratory infections, especially Newcastle disease and infectious bronchitis, may (1) activate latent Mycoplasma infection, (2) "trigger" field outbreaks of air-sac disease, (3) increase impairment of performance, and (4) cause much more severe symptoms and lesions in birds with the CRD-air sac syndrome.

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CURRENT EVALUATION OF CHRONIC RESPIRATORY DISEASE

By H. Van Roekel[1]

Chronic respiratory disease (CRD) is recognized as one of the important poultry diseases confronting the industry at present. The gravity of the losses resulting from this disease has been emphasized by the alarming condemnation rate in poultry slaughtering plants. Field and laboratory observations indicate that condemnations due to aerosacculitis may frequently result from mixed infections. However, in numerous outbreaks of aerosacculitis the disease follows a prolonged course, hence the name chronic respiratory disease.

The etiology of CRD is a pleuropneumonialike organism (PPLO) that has been designated M. gallisepticum. Other PPLO, nonpathogenic for poultry, may be encountered in the bacteriological examination of the respiratory tract of poultry. Different sero-types and cultural attributes have been described for various strains. The fastidious growth requirements of the agent have retarded the study of its cultural, biochemical, and serological properties. However, recent research findings concerning the nature and characteristics of the agent have been encouraging. Further basic information concerning the agent is needed to improve diagnostic procedures and control methods for the disease. It is hoped that research workers who have adequate and proper facilities for basic studies of the agent will accept the challenge to procure the needed knowledge.

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Our information concerning the behavior of CRD under various flock conditions is limited. Uncomplicated CRD may express itself by various behavior patterns. These patterns may be further modified by certain stress factors. To understand more clearly the natural disease in its various forms the influence of contributing factors must be investigated. Factors such as live virus vaccination, faulty ventilation, improper temperature, insanitary methods, and general flock health may exert a definite influence on the precipitation and severity of the disease.

Current methods of producing broilers are only as successful as the disease prevention procedures adopted for rearing the birds. The CRD problem that confronts the industry can be greatly lessened if producers improve their management methods. It is suggested that controlled experiments be conducted under field conditions so that results will have a practical application. Few State experiment stations have facilities to attempt a research project of this scope. It becomes apparent that the cooperation of commercial growers must be solicited to the extent that a sound research project can be undertaken.

The known host susceptibility for avian PPLO is limited. At this time it would seem most expedient to determine what other domestic and feral birds may serve as hosts in addition to the chicken, turkey, and a few other species that have been mentioned in the literature. Additional information on this phase of the problem would be particularly valuable in rearing replacement flocks for chickens and turkeys; it would be essential in an eradication program.

The different modes of transmitting CRD concern the breeder, hatcheryman, broiler grower, and commercial egg producer. Egg transmission of the disease appears to be the primary mode of disseminating the agent. The degree and duration of egg transmission may vary in different flocks or within the same flock. Flocks recently infected may transmit the organism with a greater incidence than flocks that have had the disease for many months.

The infection may also spread by direct and indirect contact. This is especially significant in adding infected birds to noninfected flocks, in rearing flocks on contaminated litter or ranges, and in establishing or maintaining CRD-negative flocks.

Investigations of the immunological aspects of the disease have revealed that agglutinating antibodies are developed in the host after infection. These agglutinating substances may be detected with specific PPLO antigen. At present only the S-6 type described by Adler is used for serological testing. Antigens prepared from this sero-type have been used in three different macroscopic agglutination tests. These antigens are prepared with considerable difficulty and may vary in their sensitivity in detecting chickens that have had contact with the agent. Use of the agglutination test for testing turkey sera has yielded disappointing results to date. In chickens this test has been helpful in establishing the disease status of the flock.

The hemagglutinating-inhibition test has also been used, but its application is limited largely to experimental projects. The serum-plate agglutination test has been very useful in measuring the CRD status of a flock. However, the test may be further improved through refinements in the preparation of the antigen and through greater elucidation of the immunity of the host.

Control of CRD has presented many problems. Considerable time, effort, and money are spent on medicating flocks to prevent outbreaks or to minimize losses from the disease. Recent investigations have demonstrated that potentiation with either teraphthalic acid or low calcium diets results in increased antibiotic blood levels. However, antibiotic potentiation does not provide an adequate solution to the problem.

Also, injection of antibiotics into day-old chicks has been practiced recently. The results of these trials have not been made available for critical evaluation.

Probably the most promising results for control of CRD have been obtained by dipping hatching eggs in an antibiotic solution. The encouraging results justify an immediate expansion in research regarding this method of control. If it should prove to have merit, it will greatly aid the industry in reducing the PPLO infection in all types of stock.

Finally, it should be emphasized that proper management and sanitation are important counterparts of any disease control or eradication program that employs measures to detect and to eliminate the disease.

THE PROBLEM OF ESCHERICHIA COLI IN AIR SAC INFECTION

By W. B. Gross[1]

Air sac disease became a serious problem after the widespread use of live virus vaccines. Recently, the inspection of poultry meat for wholesomeness has resulted in the rejection of many infected birds that did not die. Among the more common lesions are pericarditis and perihepatitis. From the pericardial sacs and livers Escherichia coli can be isolated. These isolates can be placed in 6 serotypes of which only 3 are common. Recently these types were isolated from about 50 percent of samples of feed and litter collected from feed mills and poultry farms in Virginia.

As few as 10 bacteria of the pathogenic serotype can produce pericarditis when inoculated into the air sacs of chickens. The incidence can be increased by increasing the number of bacteria or by adding media or yolk sac passed PPLO to the inoculum. India ink can be substituted for the PPLO. When inoculated IV over 10^7 bacteria are required to produce the same incidence of pericarditis as 10 inoculated into the air sac. Birds from a group that had a IBV-PPLO infection, which would allow a 50 percent incidence of pericarditis when exposed to aerosols of E. coli, were as resistant as normal birds to IV inoculation.

Birds, which have a respiratory disease due to pleuropneumonia-like organism (PPLO), infectious bronchitis (IBV) or Newcastle disease virus (NDV) alone or in combination, became susceptible to E. coli administration in an aerosol. The most important agent is PPLO which when given alone into the air sacs of chickens renders them susceptible to E. coli invasion, for about 8 to 40 days. Viruses tend to increase the spread of PPLO to contact birds and to increase the susceptibility to E. coli particularly early and late in the course of the disease. Vaccine viruses are almost as effective as field strains in making PPLO infected birds susceptible to E. coli.

From our research work the following explanation of air sac disease outbreaks is postulated. Small numbers of PPLO infected birds hatch with normal chicks. The spread of PPLO to contact chicks is favored by virus infections (field or vaccine strains), low brooding temperature, poor ventilation, and crowding. The bird centers its defense mechanism against the PPLO, IBV, and NDV. This defense is slanted strongly in the direction of the mononuclear cells, particularly lymphocytes. An effective defense against pathogenic E. coli is an early heterophile response. Since the pathogenic strains of E. coli are commonly found in the feed and litter, a source of bacteria is frequently available. Respiratory-disease-infected birds that cannot make a strong heterophile response following aerosol exposure to E. coli develop pericarditis; many die. The E. coli infection is of very short duration, requiring less than five days for pericarditis to develop or for a resistant bird to control the infection and body temperature to return to normal. It may take a month or more for the heart and liver lesions to heal even if adequate therapy is administered.

Other diseases or lesions caused by the pathogenic type, E. coli are panophthalmitis, salpingitis, peritonitis, a fowl typhoidlike disease and synovitis.

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THE SALMONELLA INFECTIONS (PULLORUM, TYPHOID, AND PARATYPHOID)

By D. E. Stover[1]

Through application of appropriate measures based on research, great advances have been made toward the eradication of pullorum disease and fowl typhoid of poultry in the United States. Pullorum disease, caused by the bacterium Salmonella pullorum, and fowl typhoid, caused by a closely related bacterium, S. gallinarum, appear to be under good control in most states. Both are ovarian transmitted and therefore are known as egg-borne diseases. They are susceptible to control and eventual eradication through application of the agglutination test to prospective breeding stock followed by bacteriological examination of reactors, the elimination of infected groups of birds, and the destruction of the infectious agent wherever it may remain on the premises.

Following any test in which infection is found, the elimination of the entire flock, or at least the elimination of entire houses or pens of birds that are infected, has generally been much more effective than the removal of only the reactors. The former procedure should be encouraged and the latter discouraged or even prohibited under an official pullorum-typhoid control program.

By far the greatest progress in the control of these two diseases has been made during the period that the National Poultry and Turkey Improvement Plans have been in operation. The annual testing of all flocks participating in the Plans has gradually decreased the incidence of pullorum disease and fowl typhoid until now it is at its lowest point. The percentage of reactors on first test of chickens in 1960 reached a new low of 0.018 and in turkeys a new low of 0.007. Unquestionably, many breeder-hatcherymen operating outside of official programs also have been highly successful in their efforts to control these diseases.

As in some of the programs designed to control other diseases of livestock and poultry, the reduction of the incidence from a fraction of 1 percent to 0.0 percent will be the most difficult, but there is reason to believe that in the case of pullorum disease and fowl typhoid it eventually will be accomplished in every State. There is much interest now in the possibility of complete eradication. If this is attempted, no doubt more drastic measures will have to be used on a nationwide basis than have been used in the past.

The Committee on Pullorum Disease Eradication of the American Association of Avian Pathologists recently has made a survey of pullorum-typhoid control programs in the United States. It would appear that industry in general prefers to keep the National Plans programs on a voluntary basis at the present time. The committee definitely believes pullorum disease and fowl typhoid can be eradicated. It has suggested that to accomplish this goal each State analyze its program and consider the following areas in the development of an all-out effort:

- The basic principles for the identification of a U. S. Pullorum-Typhoid Clean flock under the National Plans are acceptable as the starting point for an eradication program.
- A Federal regulation be adopted to control the interstate movement of poultry from the standpoint of pullorum disease and fowl typhoid. If this does not receive adequate support, then each State develop its own state regulation controlling the importation of hatching eggs and poultry from the standpoint of pullorum disease and fowl typhoid.
- All turkey and chicken breeding flocks as well as other fowl in the State be under a pullorum-typhoid control program and be classified as U. S. Pullorum-Typhoid Clean or its equivalent.
- All outbreaks of pullorum disease and fowl typhoid be reported to the proper state agency that has regulatory power and such flocks be quarantined, and the

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marketing for slaughter of such flocks be done through a plant under State or Federal supervision. Other types of Salmonella and Arizona infections be reported to the proper state agency.

- Poultry consigned to public exhibitions, such as county, state fairs, and poultry shows, originate from U. S. Pullorum-Typhoid Clean flocks or its equivalent.
- A State or area may be designated as Pullorum-Typhoid Free if all turkey and chicken breeding flocks are classified as U. S. Pullorum-Typhoid Clean or its equivalent and no flocks are under quarantine in the area.

Those importing states that have made good progress in the control of pullorum disease and fowl typhoid are the ones most liable to be adversely affected by the failure to prevent the interstate movement of infected poultry and hatching eggs. A proposed Federal regulation on the interstate movement of poultry and hatching eggs has been introduced for consideration. If finally adopted, this regulation will, next to the adoption of the National Plans, be the most important step ever taken in the attempted control of pullorum disease and fowl typhoid on a nationwide basis. A recommendation favoring the promulgation by the Animal Disease Eradication Division (ARS) of a regulation of this type was made at the National Plans Conference held on June 24-25, 1958, in Louisville, Ky. Incidentally, the National Plans were made even more effective by the approval and acceptance at its last conference on June 21-23, 1960, in Athens, Ga., of a set of auxiliary provisions which give increased emphasis to hatchery and flock sanitation procedures. Members of the Committee on Pullorum Disease Eradication of the American Association of Avian Pathologists served on a national committee which developed these procedures. The auxiliary provisions will help to strengthen the over-all Salmonella control program.

The benefits to be derived from the complete eradication of pullorum disease and fowl typhoid in the United States should not be underestimated. Economic losses that have resulted from these diseases have been very great, and the cost of testing programs has been very high. Eliminate such expenses in the future and the money saved can be used to good advantage in the control of others poultry diseases which apparently do not lend themselves to early eradication.

Paratyphoid

Following the eventual eradication of pullorum disease and fowl typhoid there will continue to be numerous other Salmonella infections in poultry to be dealt with. Members of the genus Salmonella other than those that cause pullorum disease and fowl typhoid are commonly known as the paratyphoids. Of approximately 500 Salmonella serotypes that have been identified throughout the world, more than 100 have been reported in chickens and turkeys in the United States. All are considered to be potentially pathogenic for animal life, and many of the species found in chickens and turkeys definitely have been observed to cause clinical infections. As a group, Salmonella organisms have a very wide host range.

Paratyphoid infections have generally been a more serious problem in turkeys than in other poultry. Several States have developed fairly effective Salmonella typhimurium testing services for controlling this infection in turkeys. In most areas S. typhimurium has been considered to be next in importance after S. pullorum and S. gallinarum. Its much wider host range makes it an even more serious problem in that one respect. Testing prospective turkey breeding flocks for S. typhimurium at the same time they are tested for pullorum disease and fowl typhoid has become a rather general procedure in some states. Typhimurium testing, although not required under the National Plans, has served the turkey industry in two ways. First: It has detected many infected flocks that were disposed of immediately, thus eliminating the infection in that manner. Second: It has detected many other infected flocks that were held for hatching egg production, but the owners were forewarned that very rigid sanitary measures, including early heavy fumigation of the eggs, would have to be used to prevent infection in the progeny.

Doubt has been expressed that flock testing will contribute generally to the solution of the paratyphoid problem. There are other avenues of approach which seem to look more promising. Since research has shown that Salmonella organisms other than S. pullorum and S. gallinarum are deposited on the surface of hatching eggs through fecal contamination much more often than they are incorporated within the eggs from ovarian infection,

they are more susceptible to control by application of sanitary measures.

Producing clean eggs from clean nests and transporting them in new or clean, fumigated cases will definitely help prevent the spread of paratyphoid infections. The formaldehyde-gas fumigation of turkey eggs on the farm right after they are gathered is becoming a common practice. Fumigation is then repeated at the hatchery. Very early fumigation of chicken and turkey eggs is being recommended because of numerous reports by research workers that given sufficient time Salmonella organisms from fecal contaminations are capable of entering the egg by shell penetration and then being transmitted to the progeny by this route. Fast action, using heavier fumigation than formerly recommended, may be required if the organisms are to be killed. The recently added auxiliary provisions of the National Plans (USDA Miscl. Pub. 739, revised May 1961, pp. 38-41) should be referred to for details on the correct methods of hatching egg fumigation and the cleaning and disinfecting of equipment on the farm and in the hatchery. An illustrated leaflet (Bureau of Livestock Disease Control, Division of Animal Industry, State Department of Agriculture, Sacramento, Calif.) emphasizing the importance of early heavy fumigation has been published by the California Department of Agriculture.

It is becoming increasingly evident that with the adoption of more effective sanitary procedures in the handling of hatching eggs, certain environmental factors are a continuing problem. This has been exemplified on turkey breeding farms where S. typhimurium infections have occurred in the breeding stock from year to year but have not occurred in the progeny brooded and kept for breeding purposes on other farms. It would appear that either there has not been a proper cleanup on the home farm or that the infection remains in a rodent or other wildlife population. A strong effort should be made to eliminate such carriers and to destory any of the infectious agent remaining on the premises.

On the basis of the many recent reports of Salmonella of different serotypes in poultry feeds and in animal and vegetable materials that might be incorporated in poultry feeds, it can be assumed that a serious problem has developed. While drugs are frequently prescribed for use in feeds for the purpose of combatting Salmonella infections, it is ridiculous to have to consider that the very feeds that contain these drugs may also contain an etiologic agent they are intended to combat. One of the easiest ways for poultry to become infected with Salmonella organisms is to consume contaminated feed. An immediate active outbreak may occur, or some of the birds may become carriers and transmit the infection to the progeny or other poultry, in which an active outbreak may occur. Probably under certain conditions feed contaminations never result in clinical infections in poultry.

The numerous findings of Salmonella organisms in poultry and livestock feeds by laboratory workers would definitely suggest that there should be considerable improvement in the sanitary procedures followed in some plants in the preparation and storage of feeds, particularly the animal byproducts intended for use in feeds. A task force of selected personnel from the Animal Disease Eradication Division has been designated to study and report on the problem. Requests for this review were received by the Agricultural Research Service from the North Central States Poultry Disease Conference, the Committee on Salmonellosis and Related Enteric Diseases of the National Plans Conference, the American Association of Avian Pathologists, and the United States Livestock Sanitary Association. If it is believed that immediate action is necessary, the task force will formulate proposals that will be useful to governmental agencies and the poultry and livestock industry.

It has been indicated in reports of the isolation of Salmonella organisms from feeds that certain cultural methods may be better than others for determining if feed samples are contaminated with these organisms. If bacteriological examinations of feeds are to be made routinely for any purpose, consideration should be given to using feed sampling methods and cultural techniques of proven superiority.

This discussion should not be concluded without mentioning the importance of the prompt isolation, identification, and typing of Salmonella organisms when present in poultry, their feed or their environment and the immediate reporting of these findings to the responsible disease control officials. Unless the facts become known soon after contaminations or occurrence of infections, the opportunities for preventing the dissemination of the infectious

agents are greatly reduced. The laboratory of the State Department of Public Health in several states and the Communicable Disease Center's Enteric Bacteriology Laboratory at Chamblee, Ga., have been very helpful to livestock and poultry pathology laboratories throughout the country in the typing of Salmonella cultures. Cooperative efforts such as these will help to bring the Salmonella infections under more effective control.

COCCIDIOSIS CONTROL

By W. Malcolm Reid[1]

Coccidiosis remains a very costly disease for the poultry industry. In a careful study of losses in chickens, Foster in 1949 estimated a 5 million dollar annual loss due to mortality for coccidiosis and another 5 million dollars from morbidity, drugs, disinfectants, labor loss, and other indirect costs. Since that time preventive coccidiostats have come into regular use. Pharmaceutical house estimates range from 15 to 25 million dollars per year as the domestic coccidiostat market. These figures are fairly realistic when the tonnage of feed containing a coccidiostat is calculated. Thus, the cost of coccidiostats alone now exceeds the losses estimated by Foster in 1949.

A second figure by the Agricultural Research Service of the USDA (1954) estimates losses from coccidiosis in excess of 38 million dollars per year over the 10 year period of 1942 to 1951. Actual dollar values are realistic for medication costs but might need to be lowered for morbidity and mortality because the laws of supply and demand affect price structure. Nevertheless, we must conclude that coccidiosis is still an expensive disease.

Before thinking in terms of future coccidiosis research and control programs, let us take a quick look at four different methods being used in coccidiosis control today. Presenting them in the order of their appearance, the first method, dating back to before coccidiostats were available, emphasizes sanitation. This includes proper litter management to prevent wet areas where large numbers of occysts may sporulate. Proper litter management might well be re-emphasized today with all methods since poultrymen have begun to realize that complete protection always cannot be purchased in a feedbag. The emphasis upon disinfection of the premises, which was a part of the sanitation approach, now appears to be less desirable in coccidiosis control than for other diseases. Complete sterilization may postpone eventual exposure until a time when an older, completely susceptible flock succumbs to a sudden and heavy build up of oocysts. An early, light exposure may prevent this by a gradual building up of immunity. The sanitation approach is still successfully practiced by some poultrymen while others have been forced out of business trying it.

With the introduction of sulfa compounds for therapy, treatment to combat coccidiosis was made possible. This method has the advantage of conserving drugs but the disadvantage of always bringing in the coccidiostat too late to protect all the birds in the flock. Some poultrymen continue to rely on treatment when the method is combined with good litter management.

The third method, which has had wide acceptance since 1949, is the use of a preventive coccidiostat in the feed for the first 6 to 22 weeks of the bird's life. Some 17 different coccidiostats have been introduced in quick succession in the past 12 years. Table 1 lists the ones with widest acceptance, together with the date when efficacy was first reported and when commercial use was started. These drugs differ considerably in efficacy against certain species and in their mode of

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physiological action. As they were introduced, the advantages of each were well publicized by sales representatives. Competitive pharmaceutical houses searched for the disadvantages. Disadvantages both real and imagined were pointed out to prospective buyers. The resulting confusion led to establishment of a committee to set up criteria for the evaluation of coccidiostats by the American Feed Manufacturers Association. From the informal discussions of this group, and from other sources a list of the properties of the ideal coccidiostat may be compiled as follows:

1. No residual which may be harmful in human food consumption.
2. Low in cost. $1.00 per ton of feed suggested.
3. Prevent morbidity and mortality from all pathogenic species and strains of coccidia.
4. Must not be toxic to birds of any age.
5. Must not be distasteful.
6. Must be stable and easily mixed with feed.
7. Must not be electrostatic or hygroscopic.
8. Must not impart odor or flavor to eggs or meat.
9. Must be compatible with other feed additives.
10. Must not affect production or reproduction.
11. Must have good chemical analytical methods.
12. The carrier should be registered.
13. Must not interfere with development of immunity under field conditions if used for replacement flocks.

Bonus Factors

13. Bacteriostatic, fungistatic, viruscidal, anthelmintic or properties showing efficacy against ectoparasites.
14. Growth promoting activity.

The success of the coccidiostat program is attested to by the fact that most of the nearly 2 billion broilers reared last year were started on a coccidiostat. Death loss has now reached a negligible figure. However, three disadvantages may be listed. First, it may also be assumed that the cost of this medication program will be continued indefinitely. This expense must now be regarded as something of an insurance program which spreads the risk and prevents disastrous losses at any one time. Second, morbidity losses still worry the producer. This topic is treated more fully below. Third, another difficulty quoting Ferguson (1960) is that the problem of coccidiosis in replacement flocks is not diminishing. Inhibition of coccidial life cycles in the field by the widespread use of coccidiostats prevents sufficient exposure in some cases to produce immunity in adult birds. This has provided a place for development of a fourth program--that of planned immunization.

The only commercial program of this type now being used was introduced by Edgar and King in 1952. It consists of a light infection of several species of oocysts administered at the hatchery per os or in feed or drinking water at 3 days of age. Light infections initiate immune reactions and seed the litter with oocysts which must cycle into a second or third generation before a strong immunity is developed. A preventive coccidiostat is administered during the first 6 weeks to forestall an overexposure before firm immunity has been established. Under some management conditions, such a program is being used successfully for replacement stock. Its use for broilers is more problematical since the desirability of producing an immune broiler is being debated by producers.

Since none of the four programs here outlined have proved to be 100 percent satisfactory to all poultrymen, continued research and development is called for.

Let us turn our attention to five ways in which governmental regulations affect coccidiosis control programs in the poultry industry. First, let us consider the condemnation problem. In a recent study of new flocks on farms with high condemnation histories in Georgia, Clarke, Kimsey, Morris , and Schmittle (1960) found coccidiosis lesions in 14 out of 43 flocks studied when birds were sampled on a single farm visit. At marketing, condemnation ranged from 0.3 to 42.9 percent and they did not find correlation between coccidiosis and flocks with high condemnation. Their data suggest that the type of coccidiostat or early discontinuance of a coccidiostat may be more closely correlated with high condemnation. Although coccidiosis is not in the same category as the respiratory diseases, it does constitute a frequent stress factor in birds between 4 and 9 weeks of age.

Some 13 percent of the accessions brought to the diagnostic laboratory in the Northeastern States in 1959 (Angstrom, et al.) were diagnosed as coccidiosis. About 10 percent of the total accessions of the diagnostic laboratories in Georgia (Morris, 1960) were diagnosed as having one or more species of intestinal coccidiosis. Many field service reports indicate that coccidiosis is a definite stress problem, which may later affect condemnation.

A second problem relates to coccidiosis and condemnations. In some areas mild coccidiosis has resulted in condemnations. Coccidiosis is a disease of the intestine and ceca, which are discarded. Slight to marked dehydration of the carcass may occur. Cases of extreme dehydration and anemia should be condemned. Systemic disturbances and toxemia are not characteristic of this disease. It may be assumed that as training programs through the inspection service progress, the milder lesions of coccidiosis visible through the intestinal or cecal serosa will be more readily recognized as one of the causes of slight color dfferences in carcasses. This may aid the inspector in deciding that the carcass may be safely passed as wholesome.

Three other problems are related to the regulations of the Food and Drug Administration. First, the withdrawal regulations which apply to some coccidiostats but not to others, have complicated the problem of coccidiostat selection. Some operators live in fear of coccidiosis breaks after the drug withdrawal period has started. Breaks after birds are scheduled for market create a difficult and expensive problem for the processor. One integrated operator estimated a loss of over $14,000 on 2-1/2 million birds attributed largely to this withdrawal problem during 2 months in the late summer of 1960. His present program calls for three different coccidiostats on each lot of broilers. One switch follows a prestarter period on low calcium and chlorotetracycline. The other switch occurs during the premarketing period to get on a coccidiostat not requiring withdrawal.

Second, registration problems related to the so called "Grandfather clause" have plagued the feed manufacturer in recent months. The situation which permits use of the older registered formulas containing arsenicals, but prohibits use of new combinations has led to many steps backwards in feed formulation and coccidiostat development.

Third, field testing programs necessary for final assay of a coccidiostat are now greatly hampered. Small floor-pen trials or laboratory screenings do not indicate the effectiveness of a coccidiostat which needs to be tested on millions of birds under different types of conditions. Some type of temporary permit issued by the Food and Drug Administration would be well worth exploring.

The following suggestions are made for continued investigation in the field of coccidiosis control:

1. The pharmaceutical industry should be encouraged to continue its search for better and cheaper coccidiostats. This program is already well advanced with some firms having thousands of drugs scheduled for screening while others are using a more selective approach. The possibility of a coccidiostat that is safe enough and cheap enough to protect the bird against coccidiosis through its life should not be overlooked.

2. Neutral state and federal governmental agencies should continue to evaluate coccidiosis control programs with emphasis upon field performance. With layers, more emphasis needs to be placed on relationships between coccidiostats and immunization under field conditions.

3. Encouragement should be given to further improvements in planned immunization programs. Other methods of inducing immunization may well be explored under field conditions. A search should be encouraged for non-virulent strains of coccidial species to be used in such programs.

4. A rapid method such as the flourescent antibody technique for detecting flock exposure to a given species would assist in diagnosis, chemotherapy suggestions, and in studying the relationships between coccidiostats and immunity.

5. Further studies on the distribution of various strains and species of coccidia within the United States and on a worldwide basis should be encouraged. This study should include the effects of coccidiostat use in development of resistant strains.

6. The establishment of a repository of coccidial species and strains maintained in a manner similar to that of the American Type Culture Collection has been proposed. Such a "library" would be of great assistance to all investigators working on a coccidiosis problem. The USDA at Beltsville, Ames, or Athens has been suggested as the most likely agency to direct such an activity.

Table. 1. Coccidiostats used commercially in feed for prevention of coccidiosis

Coccidiostat	Company	First commercial use	First report of efficacy in scientific literature
1. Sulfaquinoxaline	Merck	1948	Delaplane, et al., 1947
2. Nitrofurazone	Hess & Clark	1948	Harwood, et al., 1947
3. Nitrophenide	Amer. Cyanamid	1949	Waletzky, et al., 1949
4. 3-Nitro	Salsbury	1951	Morehouse and Mayfield, 1944
5. Nitrosal	Salsbury	1953	Morehouse and Mayfield, 1948
6. Arzene	Amer. Cyanamid	1953	
7. Polystat	Salsbury	1954	Zbornik, et al., 1955
8. Nicarbazin	Merck	1955	Barber, 1955
9. Bifuran	Hess & Clark	1956	Harwood, et al., 1956
10. Trithiadol	Sterwin	1957	Coulston and Dennis, 1958
11. Furazolidone	Hess & Clark	1957	Harwood and Stunz, 1954
12. Whitsyn-10	Whitmoyer	1957	Lux, 1954
13. Unistat	Salsbury	1958	Morehouse and McGuire, 1957
14. Glycamide	Merck	1958	Cuckler, et al., 1958
15. Zoalene	Dow	1960	Hymas and Stevenson, 1960
16. Amprol	Merck	1960	Cuckler, et al., 1960
17. Potentiated Aureomycin	Amer. Cyanamid	1960	Peterson, 1958

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THE AVIAN LEUKOSIS COMPLEX PROBLEM

By B. R. Burmester and T. N. Fredrickson[1]

CURRENT LYMPHOMATOSIS PICTURE

There are three main sources of information indicating the relative incidence and economic importance of a poultry disease: (1) the data compiled by the Agricultural Marketing Service for condemnations in poultry dressing plants; (2) the diagnoses of consignments in State diagnostic laboratories; and (3) the mortality data from random sample poultry tests held in various states. A brief survey of a few figures compiled from these sources will give us an idea of the relative importance of the avian leukosis complex in the poultry industry today.

In a recently completed survey of condemnation of birds from 49 flocks in the Delmarva area, Cover and Benton (communication, 1961) found that approximately 20 percent of condemnations were due to lesions caused by diseases of the leukosis complex. From their figures, which were taken from Agricultural Marketing Service records, it is apparent that leukosis is a constant but relatively unspectacular problem. In only two flocks was it responsible for more than 40 percent of condemnations, and it was never responsible for the high condemnation rate in those flocks in which 9 percent or more of the total flock was condemned. In the Delmarva area skin leukosis is a prominent problem; however, Cover and Benton (communication, 1961) found that 92 percent of the birds which had skin leukosis also had lesions of leukosis in the internal organs.

In a survey of condemnation figures for frying chickens in New Hampshire dressing plants, Dunlop (communication, 1960) found that leukosis lesions were responsible for about 5 percent of the condemnations.

We have selected figures from the State diagnostic laboratories of New Hampshire and Pennsylvania to illustrate the relative number of cases of leukosis diagnosed in these laboratories as compared to other diseases. In New Hampshire consignments with leukosis made up 20 percent of the total number in 1959. Dr. P. H. Seitz has provided us with some interesting data from the six poultry diagnostic laboratories in Pennsylvania. The percentage of consignments with a diagnosis of leukosis has increased steadily from a 13.8 percent in 1955 to 26.2 in 1960, approximately a twofold increase. Both visceral and neural forms have increased at the same rate, with visceral being about twice the neural form rate. In contrast, consignments diagnosed as chronic respiratory disease have remained almost unchanged, and in 1960 respiratory diseases were less than half the consignments with leukosis.

Mortality data from random sample tests provide useful information, because in most such tests the causes of death of all birds are ascertained by poultry pathologists and good mortality records are kept. Heavy mortality from leukosis has been reported in random sample tests in Pennsylvania, where it caused 25 percent mortality in 1960, and New York, with 7.5 percent in 1959. Wisconsin has also reported heavy leukosis-caused mortality in the random sample test.

Recent reports from poultry pathologists in several sections of the United States, and our own observations indicate that there has been a trend, within the last 2 or 3 years, toward a change in the disease picture. Visceral lymphomatosis is generally described (Jungherr and Dmochowski, Feldman and Olson, in "Poultry Diseases," Biester and Schwarte, 1959) as a disease of mature chickens, with mortality starting at the time of sexual maturity and occurring continually at a low level for many months, with terminal lesions on enlarged, pale livers and spleens. Recently leukosis has been observed in many flocks as acute outbreaks of the disease (Dank Morris in

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"Georgia Poultry Times," Sept. 1960). Mortality may start as early as 7 weeks, but more usually after 12 weeks of age, rapidly reaches a high peak, and drops to a low level in a few weeks' time. Lesions in chickens of such flocks involve the gonads, kidney, lung, heart, muscle, and skin, although the liver and spleen are also common sites of tumor development. In some areas certain sites, such as the skin, muscle or gonad, may for a time predominate.

Cases of neural lymphomatosis, with or without visceral involvement, are often seen in these acute outbreaks. Nerve lesions usually are seen at the start of the outbreak. It should be emphasized, however, that the chronic type of leukosis is still a major cause of mortality in older birds, even in those flocks which have had previously an outbreak of the more acute type.

RESEARCH PROGRESS

It has been shown, by experiments carried out at the Regional Poultry Research Laboratory, that the RPL12 tumor virus can induce visceral lymphomatosis, erythroblastosis, osteopetrosis, endothelioma, and fibrosarcoma. The particular tumor induced varies largely with the dose of virus inoculated and the age of the chicken at time of inoculation, and the route of inoculation. The genetic constitution of the chicken is no doubt also very important. The pathogenesis of visceral lymphomatosis and erythroblastosis has been intensively studied, and it appears that these two leukoses are intimately related from an etiological point of view. The relationship of the other tumors to visceral lymphomatosis is an area of active research at this time.

Repeated inoculation of large amounts of virus into the breast muscles of mature hens causes an immune response; that is, the tumor virus will grow in the hen's tissues but antibodies which neutralize the virus are elicited instead of tumors. These antibodies are passed into the egg and will give some protection to the hen's progeny. This method of immunization has so far been the one that has the best potential for the prevention of visceral lymphomatosis in producers' flocks. There are, however, many practical problems yet to be solved.

The mode of infection has been investigated at the Laboratory. By infectivity experiments, it has been found that high virus titers exist in the saliva and feces of shedder chick progeny from infected hens. Despite these high concentrations of virus it has been impossible to infect susceptible chicks by exposing them to water, feed, and floor droppings, or by circulating room air from infected chicks. The only method for successful virus transmission has been through direct contact over long periods with infected chicks. Under field conditions the mode of infection is very difficult to explain. Why, for instance, does leukosis often appear in one part of a single hatch while it does not appear in the other part of the same hatch on another farm? We have been unable to find any common factor that would explain the appearance of leukosis on the one farm and not the other.

Since 1945 the majority of the experimental work at the Regional Poultry Research Laboratory has utilized the RPL12 strain of leukosis virus. Beginning in 1954 periodic collections were made of tumor tissue, oral washings, and serum from chickens in field flocks with a high incidence of visceral lymphomatosis, neural lymphomatosis, or ocular lymphomatosis. This was done to find out if the viruses in this field material could be serially transmitted, and, if so, how the pathology, virulence, and susceptibility to genetic resistance and the immunological characteristics compared to the RPL12 virus. Field material was obtained from 22 flocks in the Northeast, Southeast, Midwest, and Pacific coast areas. At the time of collection, about half of the flocks had a high mortality of leukosis in the acute form, and the other flocks were of older birds with a lower incidence of the classical "big liver" type of leukosis. Very briefly it was found that most of the viruses from the visceral lymphomatosis material could be serially transmitted in chickens and they induced a high incidence of tumors. Material from neural and ocular lymphomatosis induced a much lower incidence of tumors. The oncogenic spectrum of all the viruses so far transmitted closely resembles that of RPL12 virus. Thus visceral lymphomatosis, erythroblastosis, endotheliomas, and fibromyxosarcomas have been observed in chickens inoculated with the field strains of viruses and it appears, therefore, that these viruses and RPL12 viruses are very closely related. So far we have been unable to induce the acute

form of lymphomatosis with any of the field strains of viruses or RPL12 virus, only the "big liver" type. Nor have we been able to induce lymphoid lesions in a particular site, e.g., skin, nerve, or eye. We have not been able to duplicate these acute outbreaks in our chickens even when we use virus from flocks with the acute form of leukosis. This lack of success may be due to: a difference in the genetic susceptibility in birds of the field flocks and our line 151 birds, a change in the virus under experimental transmission conditions, the presence of certain environmental factors present on those farms in which these acute outbreaks occur but not in our poultry plant.

The avian leukosis complex is in some respects similar to other viral poultry diseases. With leukosis, as with avian encephalomyelitis ("Calnek, B. W., Taylor, P. J., and Sevoian, M., Studies on avian encephalomyelitis. IV. Epizootiology." Avian Dis. 4: 325, 1960; see also "Olesuik, O. M. and Van Roekel, H., Transmission on Chronic Respiratory Diseases in Chickens." Avian Dis. 4: 348, 1960), hens shed virus through the egg, and these infected progeny provide the source of infection to nonimmune chicks of the same hatch, which come down with typical symptoms and develop lesions; shedder adults may exhibit no clinical signs of the disease, and the rate of infection among adults is probably very high and distribution of the virus is widespread throughout the United States and Canada.

FUTURE DEVELOPMENT IN RESEARCH

One of the greatest needs for more rapid advances in research on this disease complex is the development of serological procedures for the detection of chickens that have been infected and for a rapid determination of antibody levels. A sensitive laboratory method for the detection of virus is also needed. With these two procedures, rapid advances could be made in elucidating the modes of infection, the rate and distribution of infection, the factors affecting the spread of infection, and other important aspects of the epizootiology of the tumor virus.

This information is important not only in devising methods for controlling the spread of infection but also for the development of a procedure of control by the use of a vaccine.

Serological procedures are also necessary for determining the antigenic relationship between various lymphomatosis virus isolates and between viruses causing the different neoplasms of the chicken. It is important to know if such viruses are antigenically quite similar or distinctly different.

Epizootiologic studies carried out in the field are greatly needed. Such studies would not only give us valuable information on the continued occurrence of the disease in defined areas, but, more important, information on secondary etiologic factors and of other influencing factors such as environment, nutrition, disease, vaccination, etc., would be obtained and analyzed. This information might suggest possible leads that would bring about the identification and assessment of factors which have an important bearing on tumor development in the chicken.

Another area that needs further exploration and development is the induction of increased resistance or even immunity by the use of vaccines. We are now studying new strains of virus with the hope of obtaining one that is more antigenic, i.e., gives a stronger immune response than strain RPL12. An avirulent but highly antigenic strain is needed for research on the direct immunization of chickens.

The histogenesis of the fowl tumors, especially visceral lymphomatosis, is largely unknown. Careful microscopic studies of tumor lesions in various stages of their development must be carried out. Knowledge gained from such studies would tell us where the tumor starts to grow, the stages of growth, and how long it takes from the time of detection of tumor to death of the host. So far most of the histopathological studies have been on tumor lesions of dying or dead chickens with relatively little attention paid to the important early stages of tumor growth.

TREATMENT OF BROILERS--WHEN, WITH WHAT, AND HOW

By M. S. Cover [1]

The prevention and treatment of a disease in the flock is one of the most perplexing problems of today's broiler grower. Usually treatment means the battle against chronic respiratory disease (CRD), since it is the most prevalent malady of broilers in this country. The grower not only has the choice of preventive versus therapeutic treatments but also the choice of administering the drug in the feed, water, or environment (dust), or by injection into the individual bird.

First, let us examine the advantages and disadvantages of mass versus individual treatment methods.

By individual treatment--

(1) Each bird receives the recommended dose at a specific time.
(2) The handling of each bird produces a stress that may further injure the bird.
(3) The single dose method does not (unless repeated) provide for continuous medication for the flock.
(4) The method is expensive because of the labor and time involved.

By mass methods of treatment--

(1) Little or no stress is caused by disturbing the birds.
(2) The medication can be administered over an extended period.
(3) Only the birds that partake of the feed or water obtain medication and the dose is therefore dependent upon their appetite.
(4) The cost is less because of the ease of administration.

At this point I want to express my feelings about dust medication, fully acknowledging the glowing reports from some quarters in the field on its behalf. In my experience, based upon controlled research, I have found little to recommend dusts. For example, in one test in which high dosages of a dust-type medicament were used, only traces of it were found in the tissues of the birds although high levels were present in areas within the pen (on walls and floor). In our controlled treatment trials there has been little response to dusts. Perhaps dusting has value by reducing the contamination within the environment rather than within the sick bird.

One other point about flock treatment should be emphasized. We frequently talk glibly about the therapeutic value of a medicament (when used as a flock treatment), when actually the drug reduces the spread of the disease within the flock and does little to assist the infected bird. This fact, however, is frequently overlooked in the evaluation of certain treatments.

The most difficult question to answer concerning treatment is: Should I use a preventive-continuous type medication? This question cannot be fully answered, for the value of such a regimen depends upon the disease stress that will (in the future growing period) be placed upon the flock. It is somewhat analogous to taking an aspirin each morning because you may have a headache after breakfast. On farms and in situations where there is a history of consistent disease stress, then preventive treatment will be economically sound. However, on well-managed farms where good husbandry is used, "prevention by medication" is not usually an economic practice. In other words, antibiotics cannot replace good management. Antibiotics will, under conditions of disease stress, assist the bird to resist and also to cope with the disease agent. In areas where there is a constant disease stress, prevention by good husbandry and proper preventive medication is of value.

The treatment of a diseased flock is another question: What material should be used and by what method should it be administered? In this paper chronic respiratory disease (CRD) is used to designate the condition caused by pleuralpneumonialike organism (PPLO), while air sac is a complex syndrome with secondary infection. To understand the

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difficulty in treating air sac it is necessary to realize that it is a complicated disease syndrome. The inciting agent, PPLO, produces the initial infection, which is almost always followed, accompanied, or preceded by a secondary infection. In my opinion this secondary infection produces the greatest economic loss to the grower by bird damage and is most difficult to treat. The secondary invaders may be almost any bacterial, viral, or mycotic infection or combinations of two or more agents. Therefore, the value of any medicament is directly related to its ability to control this secondary infection. Since this may vary from flock to flock, the value of any one treatment differs. If all the trials on the treatment of air sac were examined, I doubt that any definite conclusion could be made regarding its efficacy. However, on occasion one treatment will show considerable value, in another case some other medicament will be beneficial, and in the third case both treatments will fail.

At the Delaware station (since 1952) we have tested all available medicaments for the use in air sac. Most trials have been repeated three times or more. None have shown consistent beneficial results. However, the use of a tetracycline antibiotic with low calcium potentiation has shown benefit more frequently than any other material. Potentiation should be used when a tetracycline antibiotic is being administered. The blood and tissue levels are greatly enhanced by the low calcium diet, and the value of the treatment is increased.

One additional factor has recently entered the treatment picture. This is the value of treatments in reducing the rate of condemnation. There is little controlled research on this matter from which one can formulate a conclusion. From the standpoint of flock health there are certainly certain periods in the progress of a disease through the flock when the condemnation rate would be high. In the early stages of an acute disease process many birds would be condemned for septicemia and toxemia. After this stage of the disease, fewer birds would be condemned unless an exudative or suppurative process followed. Then, condemnation under the head of inflammatory processes would occur.

Does treatment help the bird overcome these exudative and suppurative processes and thus reduce the condemnation rate? I believe that the same principle would apply here as in the treatment of air sac. If the medicament is effective against the infection(s) (secondary), then it will assist the bird in cleaning up the disease process. No one can say just how long the birds should be treated and held, but in our experience 10 to 14 days is sufficient to show some improvement if the medicament is effective.

In summary, air sac disease will continue to plague the industry until a method of reducing and eliminating the initial PPLO infection is found. The new egg dipping technique shows some promise in this direction. At the present judicious treatment by the proper use of antibiotics and other medicaments will aid the industry in reducing losses from this malady.

IMMUNIZATION PROCEDURE IN BROILERS--THEIR ROLE IN AIR SAC INFECTION AND DISEASE TRANSMISSION

By P. P. Levine [1]

Vaccination-induced stress in precipitating a disease in poultry is not completely unknown. Outbreaks of histomoniasis in young growing chickens following pox vaccination are not uncommon, particularly when the vaccine is applied at the time the birds are placed on range. In general, however, the conception of a multiple etiology in causing a disease complex has not been considered necessary until chronic respiratory disease (CRD) became a problem. Nelson's definitive work with a combination of coccobacilliform bodies and Hemophilus

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gallinarum was the first experimentally produced disease that foreshadowed our present problem.

The rediscovery and naming of the disease by Delaplane in 1943, the identification of the agent as a pleuralpneumonialike organism (PPLO) by Markham and Wong, and our belated recognition of Nelson's contribution of the 1930's are now matters of record. Transovarian transmission of PPLO announced by Van Roekel and his coworkers was a key in our understanding of the disease.

Yet some basic problems still remain only partly solved. One of these concerns the mechanism of initiation of a CRD outbreak in young birds in producers' flocks. Too often the PPLO isolates by themselves are not capable of instigating outbreaks in the laboratory comparable in severity to many of those in the field. It was clear that in the field predisposing factors were constantly present, not only to "triger" the outbreak but also to complicate the disease with other agents. Often these were obvious--implantation of diethylstilbestrol pellets, inadequate sanitation and ventilation, adventitious outbreaks of respiratory diseases due to viruses.

Prior to the advent of bronchitis vaccines, immunization by inducing a virulent infection in young stock was commonly practiced. The method was successful. From the outset, however, it had an element of risk--introducing infection in susceptible layers. As time went on, a second falling became increasingly obvious--CRD as a sequela to bronchitis immunization. Partly because of this and the widespread presence of bronchitis in broilers, the modified mild types of bronchitis and Newcastle disease vaccines were developed. It was hoped that the mild response to these vaccines would avoid the onset of CRD. Even now, the most apparent predisposing factor to the initiation of CRD still is the immunization of birds with bronchitis and Newcastle disease live virus vaccines. Indeed, the regularity with which these vaccinations were followed by CRD outbreaks forced pathologists to investigate and eliminate the possibility that the vaccines themselves were contaminated with PPLO.

Studies of field outbreaks by Sullivan et al. (4)[2] led them to conclude that virus infection as well as vaccination procedures may incite or influence the course of CRD in the field. Fahey (1) studied air sac disease in 187 flocks and found 37 cases of infectious bronchitis virus, 11 cases of Newcastle disease, and 48 cases of cecal and so called acute coccidiosis. Wasserman et al. (7), reporting on his findings with 22 cases of "air sac," found infectious bronchitis 9 times, CRD agent 2, Newcastle disease 2. Where air sac was not involved, CRD was found in 11 cases, Newcastle disease in 39, and bronchitis in 36.

Van Roekel et al. (5) gave a comprehensive and detailed report on the CRD problem. An analysis of his data on 65 field cases of CRD reveals the following:

- 4 cases - CRD associated with Newcastle disease vaccination alone.
- 4 cases - CRD associated with Newcastle disease vaccination and spontaneous or induced infectious bronchitis.
- 7 cases - CRD associated with induced or spontaneous infectious bronchitis virus. One case with no history of vaccination.
- 11 cases - CRD alsone or with coliforms. Four of these cases did not involve any vaccinations at all.
- 18 cases - CRD and Newcastle disease with or without coliforms. Six cases had no history of vaccinations.
- 21 cases - CRD, infectious bronchitis virus (natural and induced), Newcastle disease with or without coliforms. One case was involved with pox vaccinations. Two cases had no vaccinations at all.

Of the 65 cases, no vaccinations had been done in 13. All the remaining had been vaccinated with various combinations of pox, Newcastle disease, and bronchitis virus. The authors concluded that the high incidence of bronchitis might play a significant role in the occurrence and severity of CRD. "The histories and data also reveal that vaccinations with these viral agents apparently may precipitate CRD symptoms. However, CRD outbreaks have occurred where no stress factor, apparent or real, could be incriminated in the onset of the disease or the increase in the severity of the infection.

[2] Underscored figures in parentheses refer to Literature Cited at the end of this article.

Jungherr (2) in 1958 was more positive in his assertion that PPLO-infected 5-week-old chickens in the field came down with severe lesions of CRD when bronchitis was introduced 11 days after the PPLO. Strangely enough, simultaneous exposure to both agents produced no reaction when examination was done after 28 days.

Van Roekel et al. (6) in 1958 divided chicks from CRD positive dams into a stressed (bronchitis vaccine 4 weeks) and an unstressed group. In the 6 hatches, 328 unstressed chicks produced no signs and 7 with suspicious lesions, with no serology at 8 weeks. In 364 stressed birds, 16 had signs, 86 had lesions, 52 had positive serology with isolations of PPLO from 3 hatches, 2 negative and 1 questionable. In the same report mention was made of 10 commercial flocks that were serologically negative prior to dust vaccination with Newcastle disease and infectious bronchitis virus, but 8 flocks became positive after vaccination.

In a recent report (O. M. Olesiuk and Van Roekel (7) stressed 102 chicks with bronchitis originating from dams that had experienced CRD 24 weeks previously. These chicks were negative for signs, lesions, and serology. One hundred two chicks originating from dams with only 3 weeks involvment with CRD, gave positive evidence of signs, lesions, and serology to CRD.

What do all these studies signify? Evidence of CRD infection can be found in young chicks without any stress factor being supplied. Stress in the form of live virus vaccination will potentiate latent infection in young chicks. The reaction of young chickens to vaccination stress will depend among other things on whether or not they were infected per ovum. The frequency of this infection transmission in turn will vary inversely with the length of time CRD has become "stabilized" in the dams.

The type and pathogenicity of the PPLO has an important bearing on the appearance or severity of CRD in stressed birds. There are still many unknown factors involved in the causation and pathogenesis of CRD.

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SECTION IV--MANAGEMENT CONSIDERATIONS

NEGLECTED OPPORTUNITIES FOR THE GENETIC CONTROL OF POULTRY DISEASES

By F. B. Hutt[1]

My text for this talk was kindly provided by Wehr and Farr (1956)[2] when they wrote:

> "The best way to control coccidiosis of poultry is to prevent severe infections, and the best way to do that is to develop resistance to the disease."

Admittedly, those investigators were thinking more of the resistance developed from small, immunizing doses than of the genetic kind, but they would probably welcome any aid that the genes could give. In any case, I venture to appropriate their statement, literally, for my own purposes, and, as we shall see, to extend the doctrine to diseases other than coccidiosis.

In the 37 years that have elapsed since Frateur (1924) reported hereditary differences in resistance to what he called the bacillus of avian diphtheria, other investigators have shown that chickens differ genetically in ability to resist invasion by bacteria, viruses, fungi, protozoa, and parasitic worms. They differ also in ability to withstand several diseases commonly ascribed to nutritional deficiencies, but perhaps more correctly attributable to genetically abnormal requirements of certain nutritive elements. The feasibility of breeding strains of fowls that are comparatively resistant to pullorum disease, to fowl typhoid, to leucosis, or to the coccidium Eimeria tenella has been demonstrated. All this work has been reviewed elsewhere (Hutt, 1958).

To the fowl geneticist, seeing daily the great variation among individuals and families in resistance to disease, it is deplorable that the genetic resistance so clearly available is utilized so little in practice. As this valuable quality has been found in every adequate search for it, it seems probable that genetic resistance can be expected also to those diseases for which no such studies have yet been reported. Similarly, the demonstrations that genes for resistance can be accumulated by appropriate selection to breed strains resistant (separately) to bacterial infections, a viral disease, and to a protozoan parasite suggest that some of that same selection should also be effective against most other diseases--perhaps against all of them.

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[2] Figures in parentheses refer to Literature Cited at end of this article.

THE ROLE OF GENETIC RESISTANCE

What role might genetic resistance be expected to play in the actual reduction of loss from poultry diseases? When other methods of control are fully satisfactory and not too expensive, genetic resistance may be entirely unnecessary. Some people would cite pullorum disease as a case in point, but I am not one of those people. Against other diseases genetic resistance may provide the only known defense when exposure is inevitable. Thus far, that is the situation with respect to the avian leucosis complex.

Between the extremes represented by these examples come many diseases not adequately controlled by blood tests, by sulfa drugs or antibiotics, nor even by extreme devotion to sanitation. In such cases, when a flock is badly stricken, the "control" commonly recommended is to slaughter the sick, to market the healthy birds, and to start over. The happy euphemism masking this nasty business is "depopulation." This is a sad and expensive experience for the poultryman, and, biologically, when it happens to a breeding flock, it is an equally sad and expensive business for the domestic fowl.

In every flock suffering the depopulation ax, whether because of Newcastle disease, chronic respiratory disease, or other diseases, there are always some hens that have continued to lay, affected little or not at all by the bacteria or viruses that have stricken their pen mates (fig. 1). In the commercial flock their special value is not fully recognized, but in pedigreed flocks these healthy hens usually fall into certain families, with frequencies showing clearly that such birds have kept going because they have greater genetic resistance than the ones that succumb. Surely it is desirable to multiply the good genes that such resistant hens carry.

To make genetic resistance of value to the flock owner, it is not necessary to breed birds that have complete and permanent resistance to all diseases or even to one of them. Such a task would be difficult or impossible. All that is needed is to increase the proportion of the flock that can cope with a normal environment for the period of their economic usefulness. For a broiler that period is only 10 weeks or less; for layers, under present conditions, it may be 18 months at most.

As for a normal environment, we shall have to admit that for large flocks that term usually includes some ubiquitous bacteria, viruses, coccidia, and other pathogens. To many of these, most birds are resistant when other environmental influences are favorable. Under conditions of stress, however, whether from overcrowding, bad weather, poor housing, inadequate nutrition, the proportion of the flock able to cope successfully with an adverse environment and the combined attack of different organisms is considerably reduced.

It seems unlikely that the pathogens causing chronic respiratory disease, bronchitis, leucosis, synovitis, coccidiosis, and many other diseases are likely to be eradicated from the continent of North America. In tacit recognition of that fact, it is now orthodox procedure to vaccinate, when possible, against pathogens considered inescapable. Many birds--in some cases more than half the flock--have enough innate resistance to withstand the attackers without any vaccination, but for others that fortification is indispensable if the bird is to stay healthy. Against diseases for which vaccination is thus far not feasible, it is a common practice to bolster the birds' defenses with antibiotics, or even with extra vitamins.

For the commercial egg producer, these procedures are often essential if he is to stay in business. They might not be necessary if he would provide better environmental conditions and if the breeders would supply him with birds genetically better able to cope with the various kinds of stress to which they are likely to be subjected.

OPPORTUNITIES FOR BREEDING DISEASE RESISTANCE

Respiratory diseases.--It is appropriate to begin with respiratory diseases, because both this conference and the industry it serves are very much concerned about the losses that they cause.

From the evidence of Goodwin et al. (1956) it seems probable that such losses would be even greater were it not for the fact that nowadays practically all birds

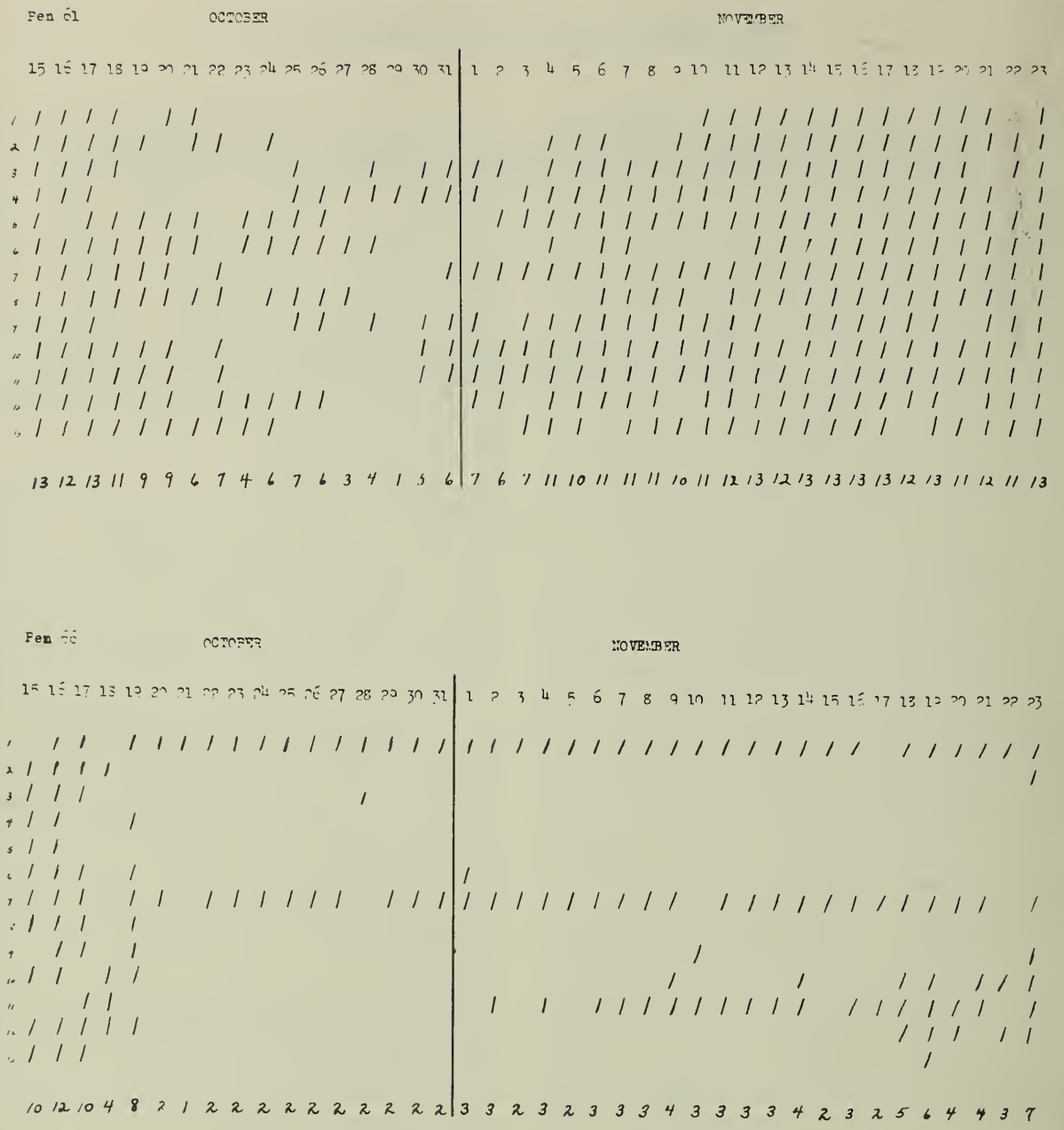

Figure 1.--Variations in resistance to Newcastle disease shown by birds of one breed in two pens stricken simultaneously at the Western New York Random Sample Laying Test. In pen 61, all 13 birds stopped laying but resumed quickly. In pen 66, most of the birds were more severely stricken, but 2 were entirely unaffected.

in large flocks are hybrids. These result mostly from crosses between different strains of White Leghorns, but some of them are crosses between breeds. In 40 different stocks of such interstrain hybrids that were severely exposed to respiratory disease, Goodwin et al. found mortality from the disease to 72 weeks of age to be consistently lower than in comparable birds of the parental pure strains that were similarly exposed at the same time. Diagnosis was based on lesions found in the respiratory tract, but no attempt was made to incriminate any one pathogen or to name the disease.

This study was conducted on such a scale that it proved beyond question the fact that hybrid vigor raises significantly the ability of laying fowls to withstand at least some form or forms of respiratory disease for 72 weeks. Hybrid vigor is genetic in origin and variable in degree.

Most large-scale poultry breeders are currently trying to find out how to get the most of this vigor for the least expense.

Here is a neglected opportunity. The program of this conference shows that respiratory disease is a major problem to the broiler industry. While most broilers are hybrids, and therefore possibly more resistant to respiratory disease than pure breeds might be, to the best of my knowledge no one has tested various crosses to determine to what extent, if any, hybrid vigor might be utilized to raise resistance to respiratory infection for the short 8 or 9 weeks in which it could be useful.

Most breeders of broilers are concerned, (1) about rate of growth, (2) about feed conversion, and (3) if at all, about resistance to disease. It is doubtful that any one of the breeders deliberately exposes his crosses to respiratory disease. Perhaps such exposure (if it can be assured) should be provided in at least one of the several broiler trials in the country. As with the deliberate exposure to leucosis in the earlier years of the New York random sample tests, the differences revealed might provide the stimulus necessary to persuade breeders of broilers that genetic resistance to disease is worth striving for. The results of such tests might first convince the buyer of broiler chicks that resistance to respiratory disease is worth paying for. Should that happen, the breeders would need little urging.

Until recently, genetic resistance in the fowl to some one specific respiratory disease had not been demonstrated, but that statement means merely that no one had made investigations adequate to reveal such differences. It has now been shown that there are clear-cut genetic differences between strains of Leghorns in ability to resist Newcastle disease (Cole and Hutt, 1961). Among young birds vaccinated on the same day with the Roakin strain of the Newcastle virus, resultant mortality was significantly higher in our strain K than in strain C, and the difference was consistent in two consecutive years. Subsequent experimental inoculation of pedigreed chicks of strain K revealed significant familial (i.e., genetic) differences in susceptibility. They indicate that ability to resist Newcastle disease could be raised in that strain by proper selection.

An objection sometimes raised against any suggestion that mortality can be reduced by breeding more resistant stock is that it would be impossible to breed a strain resistant to all diseases. It is true that genetic resistance to one disease does not carry with it ability to withstand others, but sometimes such resistance to one pathogen does raise resistance to another. Thus, breeds comparatively resistant to Salmonella pullorum are also comparatively resistant to S. gallinarum (Smith, 1956). Moreover, within one breed, strains genetically resistant to either of these bacteria excel controls in resistance to the other (Lambert, 1932).

Dr. Cole and I have now some evidence that birds genetically resistant to Newcastle disease are also more resistant to other respiratory infections as well. These last probably include chronic respiratory disease. But, because of the difficulty of incriminating separately specific pathogens, we prefer to say only that the strain of stock most resistant to Newcastle disease at about 3 months of age is also most resistant to infection of the air sacs when adult. This is a lead that should be explored further. Accordingly, we have stopped vaccinating against Newcastle disease and are hoping that we get at least enough of that each year, and of other respiratory infections as well, to shed more light on genetic resistance and to see what selection against them might accomplish.

Anyone predicting that our attempt to live with Newcastle disease is foredoomed to failure should be reminded, perhaps, that whether our Leghorns can take it or not, nothing could be a more dismal failure than the attempt to live without Newcastle disease in Great Britain. There, after 11 years of futile adherence to the policy of attempted eradication decreed by the governmental veterinarians, more birds were slaughtered in the 12th year (1959) than ever before. In the latest fiscal year for which figures have been published (1959-60), the compensation and other costs paid in the effort to eradicate Newscastle disease reached £ 4.9 million (over $13,700,000). This was nearly four times the average figure for the previous 3 years. If that campaign ever succeeds and the last skulking Newcastle virus in Britain is finally exterminated, it is to be hoped, not only that there will be an appropriate celebration in Trafalgar Square but also that another little virus of the same breed will not

be brought in next day by some innocent herring gull from across the Channel.

As we are relying in our work on natural exposure to respiratory diseases, we may be just as unlucky as Great Britain in our attempts to maintain a little pullorum disease in these same flocks. Despite the facts that agglutination tests were made on all breeding birds and that every reactor was deliberately retained in the breeding pens, pullorum disease eventually vanished, and in the past 13 years we have been able to find only one doubtful reactor.

It is to be hoped that responsibility for determining the feasibility of raising resistance to CRD and other respiratory diseases by breeding will not be left to a single laboratory. In the meantime, until some evidence is available, there seems little justification for pronouncements ex cathedra, such as a recent one concerning CRD, that "A considerable difference in genetic susceptibility must be admitted, but the answer through this approach is not a reasonable expectation" (Snoeyenbos, 1960).

Avian monocytosis.--Recommendations for control of this disease, better known to the poultryman as "blue comb," have included the administration of molasses, muriate of potash, or antibiotics. Little application, if any, has been made of the fact so clearly established by Cole (1950) and confirmed by Moultrie et al. (1955) that there are great differences among strains and among sire families in susceptibility to blue comb. In Cole's pedigreed birds, all exposed concurrently, some sires lost no daughters at all, but one male lost 47 percent of his daughters, and two of his sons both lost over 34 percent of theirs.

Unfortunately, we have no means of insuring an exposure of each generation to blue comb, and, as we have had none of it for the past 12 years (so far as we know), we have been unable to determine the feasibility of controlling it by breeding, as we have done with leucosis (Hutt and Cole, 1957). It would be helpful to breeders desiring to produce disease-resistant stock if pathologists could find some procedure for insuring adequate annual exposure to blue comb through natural channels of infection.

Synovitis.--Against this disease, currently a source of trouble to broiler growers and others, genetic resistance (to the best of my knowledge) has been neither demonstrated nor sought. Synovitis is apparently more severe in heavy breeds than in Leghorns, and with such a start, the geneticist would expect to find other signs of genetic resistance, such as differences among sire families. Inasmuch as the causative organism is not likely to be eradicated and treatments with drugs are neither cheap nor very effective, it would seem that synovitis is just the sort of disease against which genetic resistance could have considerable value. Here is a good opportunity for further research.

Encephalomalacia.--This disease is commonly attributed to destruction of vitamin E in the diet, and the prescribed remedies include the addition of supplementary alpha-tocopherol and of antioxidants to the feed to lessen destruction of vitamin E during storage. However, since in most cases the proportion of the flock affected is a minority, it would appear that when the diet is adequate for the majority, the fundamental defect is in the few birds that have abnormally high requirements of vitamin E. There is ample evidence that this weakness is genetic in origin.

As with several other conditions of stress, White Leghorns are significantly more resistant to encephalomalacia than heavy breeds (Howes and Hutt, 1952). In addition, Ranby and Outridge (1954) found among 8 sire families of about 120 chicks each that the incidence of encephalomalacia was 23 percent in one, and 12 percent in another, but in all the families from the other six sires there were only one or two cases. Such variation in sire families indicates that little selection would be necessary to eliminate the genetically defective stock.

Obviously, this is a task for the breeder of broilers--not for the grower. The grower will naturally use any supplement he can afford, if it will permit him to get more chicks to market age. If such supplements are cheap, effective, and harmless, then genetic resistance to encephalomalacia may not be necessary. Encephalomalacia should not be difficult to eliminate by breeding, because it can be easily induced by using diets high in fat, and it should be feasible to arrange uniform "exposure" at any level desired for pedigreed stock undergoing tests and selection.

Arthritis in turkeys.--A good example of a disease that depends on complex

interaction of genetic and environmental factors is provided by the arthritis that sometimes develops in turkeys at 12 to 24 weeks of age. It is almost exclusively confined to males, is greatly aggravated by rearing on slatted floors, and can be influenced slightly by modifications of the diet (Pepper and Slinger, 1955). However, the appearance of the disease shows inexplicable variations from one hatch to the next and there are remarkable differences among sire families in susceptibility to it (Johnson, 1956). Although nutritionists search for some compound to protect the biologically defective turkeys, the geneticist might well reduce their numbers by breeding, and the commercial turkey grower can avoid much of the trouble by keeping his stock (if it be susceptible) off slatted floors.

Other diseases.--The few examples considered above are perhaps adequate to illustrate the kinds of diseases against which genetic resistance could probably be utilized. Leucosis was omitted, because the value of genetic resistance to it is now generally recognized--even by people who deny that a similar defense against other diseases is possible or practicable. After steering our strains of chickens resistant to leucosis through a couple of years of cholera and steadily eliminating the more susceptible families, we have some reason to hope that they are somewhat resistant to cholera as well. At any rate we have had no known cholera for almost 20 years. Other diseases not likely to be eradicated and hence meriting some genetic study include paratyphoid infections, encephalomyelitis, and blackhead.

Kawahara (1957) found that hybrids between two breeds of chickens were significantly more resistant to blackhead than the pure parent breeds. Hybrid vigor may raise resistance to that disease, as to respiratory infections, but it is no panacea. From all indications thus far, blackhead even increases susceptibility to leucosis. This may be one reason for the occurrence of leucosis at comparatively early ages in broilers in recent years.

It has been demonstrated that resistance to Eimeria tenella can be markedly increased by selection, but I am not aware that any breeders have yet attempted to develop such genetic resistance for the birds to which it would be most useful--the chicks that go in the overcrowded pens of the broiler growers. As for laying birds, investigators having access to laying trials might well follow the ingenious lead of McGibbon (personal communication, 1960), who tested the cockeral chicks (usually discarded) at the Wisconsin Random Sample Egg Production Test with uniform dosages of E. tenella, and found significant differences among the chicks in resistance to that coccidium.

PROCEDURE

After this somewhat hortatory disquisition, it seems desirable to list the essential steps in breeding for resistance to disease. Briefly, these are:

1. Have all birds fully pedigreed as to sire and dam.
2. Insure (insofar as possible) that all birds in each generation are equally exposed to the pathogen (or nutritional deficiency) concerned.
3. Make that exposure as close as possible to the natural mode of infection, so that none of the birds' defenses are bypassed, and adequate enough to reveal familial differences in resistance but not severe enough to obliterate them.
4. Devise suitable, standard measures of resistance, such as mortality rate, age at death, and rate of growth.
5. Use family selection and progeny tests to accumulate and multiply the helpful genes of the best families.

It will be desirable in some cases, to select in two directions and thus to differentiate from the original stock two strains, one resistant and the other susceptible, but both exposed alike, as Dr. Cole and I have done with respect to leucosis. It is also necessary to show that resistance to disease can be raised without sacrificing other traits of economic value. This we have also done with respect to leucosis (Hutt, 1958).

Obviously, best results in such work are likely to be obtained by teams that include both pathologists and geneticists.

One of the greatest difficulties lies in providing a standard dosage of the pathogen. Although it is comparatively easy in some cases (coccidia), it is still completely impossible in others (leucosis). Even with dependable old friends like Salmonella pullorum, cultures become attenuated, and it is difficult to insure uniform exposure of successive generations. It will be desirable in most cases to establish the cause of death by competent diagnosis. If any pathologist can be interested in breeding for resistance to disease, he will have plenty to do.

Finally, one more neglected opportunity should be mentioned--the search for tests or indicators of any kind that might facilitate the identification of resistant animals at early ages, or even without any exposure to disease. Resistance of chicks to S. pullorum is associated with high average temperature at 1 to 6 days of age. Recently it has been demonstrated that by breeding from birds with comparatively high temperatures at that age, one soon develops a strain which, when challenged, is significantly more resistant to pullorum disease than a strain selected for early low temperature (Hutt and Crawford, 1960). Another indicator of ability to resist disease is the ring of circumocular pigment that is associated with resistance to cancer of the eyelids in Hereford cattle (Bonsma, 1949; Anderson, 1960).

Perhaps there are other similar indicators waiting to be discovered. It would be helpful to find one of these associated with resistance to leucosis, so that it would no longer be necessary for breeders to wait 18 months to identify the resistant birds. Here is a neglected opportunity for some ambitious scientist--preferably a young one.

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NUTRITION AND ITS RELATION TO DISEASE

By C. H. Hill[1]

The relationship of diet to disease has intrigued philosophers and scientists for many centuries. It seems logical when these two great environmental forces collide within a host that one would modify the other. This is the premise that underlies all work on the relationship of nutrition and disease.

When the nutritionist embarks on a study of this relationship, he quickly becomes aware that nature has beset his way with booby traps, with darkness, with the existence of nutritional entities of which he has been ignorant. Nevertheless, the nutritionist must begin sometime, and so the task has been started.

To begin with, we must recognize that all experiences relating nutrition and disease are within a definite genetic framework. The experiments of Schneider (7)[2] have indicated that a high degree of homozygosity with respect to

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[2]Figures in parentheses refer to Literature Cited at end of this article.

resistance or susceptibility prevents dietary manipulation of either of these qualities. Only in the heterozygous host does nutrition appear to influence disease. Most workers in the field of nutrition and disease ignore this. That the result is not disastrous to their experiments lies largely in the fact that most experimental animals are, in fact, heterozygous with respect to resistance and susceptibility.

We have been interested for a number of years in the effect of diet on Salmonella gallinarum infection in chicks. I should like to describe to you some of our experiences in this field to show you the paths that this research can take.

We became interested some years ago in the effect of vitamins on this disease in chicks. The vitamin requirements for growth are fairly well established, and this is taken to be the vitamin requirements of the growing chick. On the other hand, these requirements were established with healthy animals. We asked ourselves if the outcome of infection instead of growth, were the criterion, would the established requirements still be valid. To test this, we fed the chicks a corn-soybean meal type of diet supplemented with all the vitamins known to be required by the chick. The supplement and the vitamin content of the diet as well as the requirement are presented in the table 1. The dietary vitamin requirements are met by this basal diet.

To determine whether more vitamins might influence the outcome of the disease, groups of chicks were fed the same basal diet except that the vitamin supplement was increased to ten times that of the control. In this group, ascorbic acid was added at 0.1 percent. After 4 weeks on the two vitamin levels, the chicks were inoculated with Salmonella gallinarum, and the disease was allowed to run its course. Results of the first study are presented in table 2. Only 10 percent of the chicks fed the basal diet survived, while 40 percent of those fed the higher vitamin level survived. The outcome of this experiment indicated that the vitamin requirement of the chicks was increased when the response to this infection was the criterion of judgement. The high levels of vitamins, then, act as a resistance influence.

TABLE 1.--Vitamin content of the basal diet

Vitamin	Supplement/lb.	Total
Thiamine (mg.)	0.9	2.1
Riboflavin (mg.)	1.6	2.5
Pantothenic acid (mg.)	5.0	8.4
Niacin (mg.)	8.0	17.0
Folic acid (mg.)	0.45	1.55
Biotin (mcg.)	45	81
Pyridoxine (mg.)	1.6	8.7
Choline (mg.)	700	1460
Vitamin B_{12} (mcg.)	3	3
Vitamin A (I.U.)	3000	4100
Vitamin D (I.U.)	180	180
Menadione (mg.)	10	10
Vitamin E (mg.)	10	10

TABLE 2.--Influence of excess vitamins on resistance of chicks to fowl typhoid

Diet	Percent total survival[1]
Basal	10
10 X vitamins + C, 0.1 percent	40

[1] Survival time calculated from day of inoculation till day of last death in experiment.

In order that you may be clear about my terminology, I should like to say that it is my belief that resistance and susceptibility are two separate properties of a host. That is to say, an animal can be susceptible but also resistant. For operational purposes, if I add a nutrient to the diet of an infected host and the result of that addition is favorable for the host, that nutrient I call a resistance factor. On the other hand, if the addition of the nutrient is unfavorable for the host's survival, I call it a susceptibility factor. This terminology is the same as that adopted by the group (7) at the Rockefeller Institute.

We were next interested in determining whether or not the effect we observed with high levels of vitamins was due to a single vitamin or to a combination. The vitamins were arbitrarily split up into the water-soluble B vitamins, the fat-soluble vitamins, and ascorbic acid. These were fed at the same levels as that of the high vitamin supplement. The result of that study are presented in

Table 3. Only the complete supplement afforded any protection against this infection. This indicated that the effect of the high vitamin supplement was not due to a single vitamin but to a combination of vitamins.

TABLE 3.--Influence of excess vitamins on resistance of chicks to fowl typhoid

Diet	Percent total survival[1]
Basal	48
10 X vitamins + C, 0.1 percent	76
0.1 percent vitamin C	34
ADEK	15
Water-soluble vitamins	25

[1] Survival time calculated from day of inoculation till day of last death in experiment. Experimental period--17 days.

To study the role of individual vitamins further, one vitamin at a time was left out of the high vitamin mixture and the effect of this omission on the resistance of the chicks to fowl typhoid was studied. The results of that study are summarized in Table 4. The figures in parentheses indicate the multiple of the requirements that was present in the basal diet and in the high vitamin level diets when these vitamins were omitted from the high vitamin supplement. Vitamin K, for instance, was present in approximately 50 times the requirement in the basal diet; this was not enough. On the other hand, pantothenic acid was present in about twice the requirement; this was enough.

TABLE 4.--Effect of omission of individual vitamins from high level vitamin supplement on resistance to fowl typhoid

Lower resistance		No effect	
Vitamin A	(3.4)[1]	Thiamine	(1.6)[1]
Vitamin D	(2.0)	Biotin	(2.0)
Vitamin E	(--)	Folic Acid	(6.2)
Vitamin K	(50)	Pantothenic Acid	(2.0)
Vitamin B	(0.75)	Choline	(2.4)
Pyridoxine	(6.7)		
Riboflavin	(1.9)		
Niacin	(1.4)		
Ascorbic Acid	(--)		

[1] Figures in parentheses indicate multiples of requirements present in the basal diet.

These findings indicate that when the response to this infection is the criterion, the vitamin requirements are increased. But there is a certain specificity about amount of the increase for individual vitamins. It is not just a question of multiplying the requirement of all vitamins by some constant.

Another aspect of the disease-nutrition interrelationship--that we became interested in--was the effect of various levels of protein on the outcome of infection with S. gallinarum. In these experiments we employed a soybean meal-cerelose diet properly supplemented with vitamins and minerals. All the protein came from soybean meal supplemented with methionine. Throughout these studies we employed three levels of protein--10 percent, 20 percent, and 30 percent. This represents a deficiency, a sufficiency, and an excess of protein for chicks. The results of our first study are presented in Table 5.

TABLE 5.--Protein level and death of chicks from S. gallinarum

Percent protein	Days past inoculation		
	5	9	13
	Percent cumulative deaths		
10	2.9	52.9	85.3
20	12.0	72.0	93.0
30	24.0	100.0	100.0
LSD 0.05		17.7	33.5
LSD 0.01		26.8	50.7

To our surprise, as the protein level increased the mortality increased during the early stages of infection. By the end of the experimental period, however, there was no significant difference between the groups.

In this experiment the protein was added at the expense of cerelose. This resulted in an altered caloric content of the diet. In order to determine if this had any effect, the experiment was repeated and in one series the caloric content was adjusted by the use of a cellulose product. Results of that study are presented in Table 6. Results of the first study were confirmed; adjusting the caloric content of the diet did not affect the results.

TABLE 6.--Protein level, productive energy, and death of chicks from S. gallinarum

Percent protein		Productive energy	Days past inoculation		
			9	11	13
		Cal/lb.	*Percent cumulative deaths*		
10		1073	45.9	62.1	72.9
10		727	42.0	65.7	84.2
20		896	57.9	78.9	86.8
20		727	63.1	78.9	86.8
30		727	74.2	79.2	87.0
LSD 0.05	20 percent vs, 10 percent		15.3	22.4	30.8
LSD 0.01			24.0	35.1	48.3
LSD 0.05	30 percent vs. other		18.7	27.4	37.7
LSD 0.01			29.4	42.9	59.1

One other possibility that we considered was that there was something aside from protein in soybean meal which influenced the outcome of this infection in this manner. To test this, both casein and soybean meal were tested at two levels. The results of that experiment are presented in Table 7. The outcome of the infection was the same with both sources. These findings, then, indicate that protein acts as a susceptibility factor with this host and this organism.

While these findings are evidently valid under these experimental conditions, they are not universally true. Dubos and Schaedler (4, 8) have found that the effect of protein on the outcome of infections in the mouse is opposite of our findings with the chick. This means that the problems of one working in this field are extremely complex. One cannot transpose the results from one host to another even qualitatively. It is probable that the results with one organism cannot be transposed to another.

There is another caution in assigning a susceptibility role to protein in the case of chicks, and a resistance role in the case of mice. As I have pointed out, the effect in chicks came during the early stages of the infection. At the end of the experimental period the levels of protein differed little. This was also true of the work of Dubos and Schaedler. In other words, the ultimate outcome of the infection was the same, regardless of protein level fed. Schneider (6) in discussing results of this kind pointed out that nutrients, which affect the rate of mortality, act in the manner of a catalyst. The ultimate equilibrium between the living and the dead is not affected, but the rate at which the equilibrium is reached is changed. The question is then raised, "Do these nutrients really affect resistance?" We cannot be sure, because

TABLE 7.--Level of two proteins and death of chicks from S. gallinarum

Percent protein	Source	Days past inoculation		
		7	9	13
		Percent cumulative deaths		
20	Casein	29.6	66.7	66.7
30	Casein	57.6	92.3	92.3
20	Soybean	30.3	53.5	60.7
30	Soybean	56.2	77.0	79.1
LSD 0.05		12.5	25.1	24.0
LSD 0.01		18.2	36.6	32.1

we do not know the fundamental basis for resistance or susceptibility.

Studies have been carried out by L. Joe Berry (1, 2, 3) and Gilfillan et al. (5) on some basic aspects of susceptibility. These workers have shown that when animals are infected with Salmonella organisms, the citric acid content of the tissues increases. They also found that when the citric acid cycle was blocked by various inhibitors so that citric acid accumulated in the tissues, the animals died faster when infected by these organisms. The administration of citric acid or succinate, intermediates which accumulate when the cycle is blocked, also reduced survival time.

We became interested in this aspect of infection with Salmonella gallinarum and in confirmation with the work previously cited found that in this infection in chicks the blood citric acid level was increased. This increase, however, occurred just at the time the chicks began to eat less feed, a characteristic symptom of the disease. If we withheld the feed from the infected and control birds for the same length of time, there was no difference in the level of this metabolite in the blood. The results of these experiments are summarized in Table 8. In the first experiment the chicks were fed ad libitum. Five days after infection the blood was drawn for citric acid analysis. The infected chicks had significantly higher levels. In the second experiment the chicks were fed ad libitum, but at 5 days after infection feed was withdrawn from both groups for 18 hours. Blood was then collected and analyzed for citric acid. There was no significant difference between the groups under these conditions. The control group in the second experiment had blood citric acid levels similar to the infected group in the first experiment. From these data, we concluded that the increase in citric acid level of the blood in this infection could be attributed largely to the decreased feed consumption.

TABLE 8.--Blood citric acid levels of chicks infected with S. gallinarum

Items	Experiment 1	Experiment 2
	mcg/ml blood + S. D.	
Control	33.7 ± 1.7	50.3 ± 2.4
Infected	48.2 ± 3.4	52.4 ± 2.6

The problem then remained as to why decreased food consumption resulted in higher levels of citric acid. Subsequently, we found that such dissimilar conditions as changing the environmental temperatures to either 40° C. or 4° C. also resulted in an increased level of blood citric acid. These findings suggested that the increase was due to some general reaction to a stress stimulus. Since this stimulus was probably mediated through the anterior pituitary-adrenal cortex interaction, we administered ACTH both intramuscularly and intravenously and observed the effect on blood citric acid. The results of that study are presented in Table 9. Ten units of ACTH administered in either fashion resulted in a higher level of citric acid in the blood.

TABLE 9.--Effect of ACTH on citric acid concentration in chick blood [1]

Item	mcg. citric acid ml + S. E.
Control (saline)	57.8 ± 4.5
ACTH, intramuscularly	94.5 ± 7.9**
ACTH, intravenously	91.3 ± 4.6**

[1] Each observation represents an arithmetic mean for 10 birds.

**Significant at 1 percent level.

Once we had established this relationship between ACTH and blood citric acid, we were anxious to determine if the relationship might be modified by dietary manipulations. In particular, we were interested to determine whether or not the dietary changes such as increasing vitamin levels or increasing protein levels, which we found influenced the course of infection with Salmonella gallinarum, would modify the citric acid response to ACTH.

The first study was to determine the effect of increasing protein levels on the response of blood citric acid levels to ACTH administration. Results of that study are presented in Table 10. The increase in citric acid concentration as a result of ACTH administration depended upon a level of protein higher than 10 percent. This might be interpreted to indicate that under stress stimulation the chicks fed the higher protein levels responded in such a manner as far as citric acid concentration is concerned, so that they became

TABLE 10.--Effect of protein and ACTH on blood citric acid levels

Percent protein	Hours after ACTH[1] administration	
	0	6
	mcg. citric acid/ml. blood ± S.E.[2]	
10	39 ± 4.2	43 ± 2.9
20	31 ± 2.0	49 ± 2.4
30	39 ± 2.4	57 ± 4.2

[1] 4 units ACTH (Upjohn) given intravenously in 4 injections at 20 minute intervals.
[2] 5 chicks per treatment.

more susceptible to the infection, since the work of others indicated that higher levels of tissue citric acid has this influence.

Unfortunately for such a theory, when we examined the effect of protein level on the blood citric acid response to starvation we obtained the results shown in Table 11. The highest protein level actually delayed and depressed the increase in blood citric acid concentration. In theory, if the rise in blood citric acid due to anorexia which accompanies Salmonella gallinarum infection caused the chicks to die faster, the chicks fed the higher protein levels should have survived longer. Since the opposite was true, the theory must be considered invalid.

TABLE 11.--Effect of protein and starvation on blood citric acid levels[1]

Percent protein	Hours of starvation		
	0	12	36
	mcg citric acid/ml blood ± S. E.		
10	33 ± 1.8	51 ± 2.6	166 ± 8.7
20	34 ± 2.9	56 ± 9.4	153 ± 19.3
30	35 ± 3.5	38 ± 3.4	73 ± 4.1

[1] 10 chicks per group.

An examination of the effect of high vitamin levels on the citric acid response to ACTH and starvation indicates the same thing. As is indicated in Table 12, supplementing the diet with high levels of vitamins resulted in a greater citric acid response to ACTH. Starvation elicited the same response as is indicated in Table 13.

TABLE 12.--Vitamin level and citric acid response to ACTH[1]

Vitamin level	Hours after first ACTH injection	
	2	4
	citric acid μg./ml. ± S. E.[2]	
Normal	24.6 ± 2.1	27.2 ± 4.0
High[3]	34.2 ± 1.8	44.8 ± 4.9*

[1] Total of 3 units ACTH given in 4 injections at 20 minute intervals.
[2] Mean of 5 chicks per treatment.
[3] Tenfold normal vitamin supplementation plus 0.1% ascorbic acid.
*Significantly higher ($P < .05$) than at 2 hours.

TABLE 13.--Vitamin level and citric acid response to starvation

Vitamin level	Hours of starvation	
	0	20
	citric acid μg./ml. ± S. E.[1]	
Normal	29.1 ± 2.7	68.5 ± 7.9
High[2]	36.7 ± 1.8	129.4 ± 7.2**

[1] 10 chicks per treatment.
[2] Tenfold normal vitamin supplementation plus 0.1 percent ascorbic acid.
**Significantly greater ($P < .01$) than normal vitamin group.

The results of all these studies with citric acid, while important as far as the nutritional effect on various physiological and biochemical properties of the chick are concerned, leave us as far from our original goal as ever. We still do not understand the mechanism which relates these nutrients to the infection studied.

These experiences indicate that nutrition can influence disease. The extent or the direction of this influence is not yet predictable, however. The tactics of the nutritionist in this field must be to constantly probe and critically test. The goal of the nutritionist is to take his place beside the pharmacologist and immunologist in the control of disease. Such a goal is not easily won but it is worth striving for.

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PHYSIOLOGICAL RESPONSES TO MANAGEMENT

By Paul D. Sturkie[1]

A great deal is said these days about environmental physiology and the physiological responses of animals to different environments and management conditions. Unfortunately relatively little is known about it and this is particularly true regarding birds. However, since most animals, including man, exist in a changing environment, all physiology, in the final analysis, is environmental or deals with responses to these changes. An environmental physiologist is usually one who is primarily interested in the physiological responses to stressful stimuli or drastic changes in the environment, and whether the animal can adapt physiologically and even the physiological consequences of this adaptation.

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What is stress? A physiologist's definition is, "stress evokes all the physiological processes employed by the organism in attempting to maintain a steady and constant state of function (homeostasis) under unfavorable environmental conditions, both external and internal." Thus, in the attempt to maintain a steady state, following exposure to stressful stimuli, the animal evokes certain physiological responses designed to resist the stress, or adapt to it. It is well known that the particular response evoked depends upon the type of stress, and that in certain stressful stimuli, the mechanisms of the defense evoked in the process of adaptation may themselves so alter the physiological responses of the animal that, after the stress is removed, irrevocable-harmful changes may have been produced.

This is not true, however, of all stressful stimuli. In some cases, the animal may adapt without producing such deleterious changes in the body.

SEASONAL EFFECT ON BLOOD PRESSURE AND CARDIAC OUTPUT

We have known for several years that blood pressure tends to decrease in the beginning of warm weather. We have recently studied also the effects of season and changes in temperatures on cardiac output (table 1). The results show that the cardiac output and blood pressure are significantly higher in February than in August. Increasing the room temperature from 70° to 95° F. decreased cardiac output and blood pressure significantly. The decrease amounts to 35 percent.

TABLE 1.--The effects of changes in environmental temperature and season on cardiac output and blood pressure

Time and temperature	Birds	Body wt.	Mean BP[1]	Heart rate	C.O. bird	ml./m. kg.	TPRU bird	Ht.
	No.	*Kg.*	*mm./hg.*					*Jn.*
				MALES				
Aug.....	16	2.9	177	289	359	135	0.53	41
Feb.....	20	2.6	181	303	444	173	.43	45
				FEMALES				
Aug.....	15	1.96	147	347	234	121	.59	28
Feb.....	19	1.95	153	336	345	181	.46	32
Feb.....	12	1.7	147	346	481	279	.34	32
95° F.....	6	1.6	127	322	262	163	.57	31
70° F.....	[2] 6	1.7	144	356	402	240	.37	26

[1] Blood pressure.
[2] 4 of these were at 95° F.

RELATIONSHIP OF BLOOD PRESSURE LEVEL TO MORTALITY AND RESISTANCE TO STRESS

Sturkie, Ringer, and Weiss (5)[2] demonstrated a relationship between level of systolic blood pressure of chickens and mortality up to 18 months of age. The deaths during this period were significantly higher in birds whose pressures were below the mean, based upon birds

[2] Figures in parentheses refer to Literature Cited at end of article.

hatched in 1950, 1952, and 1954. We have also studied this relationship in subsequent years (1955-1959) and have developed by breeding, hypotensive and hypertensive strains (6). In the more recent studies mortality was not always higher in the lower pressure birds, and in some years there was little or no association of mortality and pressure level, particularly in years when total mortality was moderately low. Thus, the differences in viability appear to be related to general level of mortality and suggest that birds with high pressure (above average) are better able to withstand greater disease exposure and physical stresses.

To test this premise, birds with high- and low-blood pressures were subjected to high and low temperature and to physical exertion (exercise). This report concerns the results of such tests.

PROCEDURE

Healthy adult White Leghorn males and females (18 to 24 months of age) with high and low systolic blood pressures were subjected to an ambient temperature of 40.5° C. and a relative humidity of 70 until death. Pairs of high- and low-pressure birds were exposed at a given time in a small chamber.

Hypothermia and ultimate death were produced by immersing paired high- and low-pressure birds up to the neck in water at 20° C. Ambient temperatures ranged from 21° to 28° C.

Physical exertion was produced by forcing the birds to walk on a treadmill, and also in a revolving wheel. The wheel (0.6 meter in diameter) was used successfully in females but was not large enough for males. Later, the wheel was abandoned in favor of the treadmill where a horizontal belt was made to move at varying speeds by means of a variable speed motor. The birds were walked until they became exhausted and could not stand.

Systolic blood pressures were determined to an indirect technique previously described (3, 4) at 7 to 10 months of age and again just before subjecting them to the tests at 18 to 24 months of age.

RESULTS

Exposure To Low Temperatures

The results are presented in Table 2. The survival times of the high-pressure males and females were significantly greater than that of the low-pressure birds. The lethal body temperatures of the males and the high-pressure females were not different and of the same magnitude as reported previously (4), but the low-pressure females had a higher lethal temperature. This is a reflection of the lower survival time, and the more adverse effects of the treatment on the respiratory behavior of the low-pressure females, who in the earlier stages of hypothermia exhibited more severe respiratory difficulties such as gasping and cyanosis of comb.

High Temperature

Among the 11 each of high- and low-pressure females and 15 each of these groups exposed to high temperatures (40.5° C.), there were no significant differences in survival times.

Fatigue

The results are presented in Table 3. In both groups of females (A and B) the low-pressure birds became exhausted before the high-pressure ones; the difference was statistically significant.

Both high- and low-pressure males were subjected to exercise on the treadmill moving at 121 feet per minute (much higher rate than for females); there were no

TABLE 2.--Response of high- and low-blood pressure (BP) chickens to hypothermia (water 20° C.)

Item	Male			Female		
	High BP	Low BP	Pooled standard error	High BP	Low BP	Pooled standard error
No. birds.....	15	15	--	15	15	--
Body wt. (g.).....	2,423	2,410	73.0	2,050	1,903	73.0
Systolic BP (mm./hg.).....	** 241	176	4.8	** 193	131	3.0
Starting BT (° C.).....	41.2	41.3	.09	41.2	40.9	.12
BT at death (° F.).....	23.6	24.2	.36	23.5	26.5	.96
Survival Time (min.).....	** 156	113	10.1	** 115	71	9.5

*Significantly different 0.05 level.
**Significantly different 0.01 level.

TABLE 3.--Response of high- and low-blood pressure (BP) females to exercise. Treatment A, treadmill moving at 70 feet per minute and B, 12 revolutions per minute in wheel

Blood pressure	No. birds	Systolic BP	Body wt. gms.	Minute to exhaustion	Difference minute	Standard error difference
			Females A:			
High.....	14	180	1,886	37	* 11	5.3
Low.....	14	** 118	1,847	26		--
			Females B:			
High.....	20	176	1,878	26	* 8.0	2.3
Low.....	20	** 132	1,910	18	--	--

*Statistically significant at 0.05 level.
**Statistically significant at 0.01 level.

significant differences in their responses. The time required for exhaustion at this higher speed was 64 minutes, a figure considerably greater than that for the females. Thus, it is clear that males are more resistant to fatigue than females, as was shown by Garren and Shaffner (2). Whether or not high- and low-pressure males respond differently in more severe exercise remains to be determined.

DISCUSSION

The results of this study show that birds with high systolic blood pressure are better able to withstand low temperature and severe exercise than low-pressure birds. They also suggest that the difference in mortality of the high- and low-pressure individuals under natural conditions (5) is influenced by the severity of physical stresses and degree of exposure to diseases. The high- and low-pressure birds selected for these experiments were over 2 years of age and the mortality of the high and low pressure birds during that

period under natural conditions was not different.

Our earlier report (5) showed that the causes of the higher mortality in the low-pressure birds were unknown and that the distribution of diseases and disorders among the pressure groups were similar. The incidence of any one disease contributing to the total mortality was relatively low. However, it is a well known fact that routine autopsies by poultry pathologists fail to account for an appreciable number of chicken deaths. We have shown previously that the low-pressure birds are not morbid. Their body weights and rate of egg laying (Tables 2 and 3) were the same as the high-pressure birds. Observations made in years when the incidence of mild respiratory disorders was relatively high suggested a greater resistance by the high-pressure birds. However, no controlled studies on the role of blood pressure in the resistance to specific diseases have been made and are contemplated.

Everitt (1) has reported that high-blood pressure is also associated with greater life expectancy in rats and that the incidence of pulmonary diseases was higher in the low-pressure animals. Thus, the relationship between level of systolic blood pressure and life expectancy in chickens and rats appears to differ from that in man. Everitt stated that "this observation would seem contrary to human life insurance experience where hypotension is commonly associated with a reduced mortality. It must be realized that the beneficial action of hypotension on life expectancy is only true in the absence of disease," and he cites literature on this point.

What then are the physiological advantages of high-blood pressure? To quote Pickering (3), "it may enable the animal to quickly raise the blood flow to an active organ through local vasodilatation, and animals with a higher blood pressure may therefore have a wider range of organ activity. If there is any interference with the circulation to a particular organ, a higher arterial pressure may be necessary to preserve the correct conditions of capillary exchange in that organ."

CONCLUSIONS

Adult White Leghorn males and females were subjected to low temperature, high temperature, and to exercise.

There were no significant differences in the response of the high and low pressure birds to high temperature.

When hypothermia was induced by placing birds in water at 20° C., the survival times of the high pressure males and females were significantly greater than for the low pressure ones.

When high- and low-pressure birds were made to exercise (walking on treadmill) the resistance to fatigue was significantly greater in the high-pressure females but no differences were observed between the males. Both high- and low-pressure males exhibited considerably greater resistance to fatigue than females.

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HUSBANDRY PRACTICES IN RELATION TO POULTRY CONDEMNATIONS

By R. T. Parkhurst[1]

During the latter part of 1959 and the early months of 1960, I spent a lot of time in the field with broiler growers, hatcheries, feed dealers, processors, and service men. Some of you who are from the South remember the kind of winter and early spring we had down there. We had more snow in Roanoke, Va., than they did in Boston, Mass. I can recall very vividly during a visit to a processor at that time being asked by him, "Why is it that the condemnation rate is practically nil with certain growers and consistently high with others?" Unfortunately, I was not able to give him all the answers at that time; nor is it possible for me to do so today.

Husbandry practices affect condemnations to the extent that they affect the presence or absence of diseases, the control of environment, and directly determine the economic return of the broiler project. High economic returns are principally due to good management but one can be amazed at times at the good results that are obtained, in the absence of disease, with what are generally considered to be poor management practices. It is estimated that 75 to 80 percent of all the condemnations are from birds processed from only 10 percent of the growers. Surveys seem to indicate that growers who keep no records or a poor set of records are often in that 10 percent with the highest condemnations.

It is quite obvious from preceding discussions that most condemnations are due to conditions arising from respiratory diseases and air sac infections. First, there is some contamination. This is followed by a combination of disease, environmental and management conditions to which the broiler can not adapt. There is a great deal we do not know at the present time about this combination of factors.

Condemnations tend to be lower in those areas where they are charged back to the grower. I believe this is significant.

The mental attitude of the poultryman is probably the most important factor in determining whether or not any of the present programs substantially reduce condemnations. Most poultrymen do a better job of raising broilers if they have a cash incentive. The will to do a good job must be there. For that reason, the problem of condemnations is tied up with many seemingly unrelated matters.

The general opinion exists that independent growers have the lowest rate of condemnations. I do not believe that this is necessarily so. I think a good poultryman with a good incentive contract will do just as well. I am happy to say that there are still many poultrymen that do their best, irrespective of the profit motive, just because they get satisfaction out of doing a job well. However, I believe there is often a relation between the price of broilers and the rate of condemnations. When the price of broilers drops to a point that returns are marginal or there is a loss, management practices tend to

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deteriorate rapidly just when it is most important that they be the best possible.

There are many good management programs for helping reduce losses in broilers. They are usually a part of a coordinated program which also includes disease prevention and control and environmental control. I do highly recommend for your consideration the "Security Management" program of the Virginia Polytechnic Institute. An important part of this program is to keep disease out of the flock. The attitude of the poultryman is important in doing this. In my opinion, signs warning callers to keep out of the poultry house are not enough. Every broiler house should be kept locked. Hatchery and feed salesmen, hatchery, feed and operator service men and especially those who hold the contract, whether owner, hatchery, feedman or processor, should set the example. They are too often the worst offenders. Any one of them will hold the poultryman in higher regard if he insists that nobody enter a house containing broiler chicks unless he has at least met the minimum precautions in respect to disease transmission. I believe the minimum is the use of boots or rubbers, which are cleaned and disinfected before any broiler farm is visited.

Chick quality is an important factor in condemnations. When chicks are scarce and higher in price, eggs that are too small, too old or are from inferior stock are often set for hatching broiler chicks. The resulting chicks are often small, lack vigor, and grow slowly. Inferior stock would not be used for breeding when the supply of hatching eggs is adequate. Runts will be present in a flock from poor quality chicks no matter how good the management. A good poultryman will watch a bunch of poor chicks more carefully than he will good ones because he knows that usually more heat and attention are needed to keep them comfortable and free from trouble. Other growers, when they get poor chicks, give up in disgust and serious trouble usually results.

The best possible sanitation program is of little value unless the poultryman puts it into use. At the time broilers go to market, all birds should leave the premises. This not only includes mature broilers but adult chickens, bantams or game birds. They are all possible sources of infection for the next brood. After a bunch of broilers goes to market, and before the next brood comes in, the house may or may not be cleaned out thoroughly. There are those who strongly believe that, provided there has been no trouble in the previous brood and all the lumpy and wet litter is removed, the old litter can be used continuously until there is trouble. Others limit its reuse to just one time. Of course, when old litter is used, new litter is added in the area to which the baby chicks are confined. It is my observation that quite often the determining factor is the incentive that exists at the time. If the price of broilers is high, the proper sanitation procedure is followed. If the price of broilers is below or close to the cost of production, the old litter is used. Sometimes, the presence or absence of disease or parasites is the minor consideration. The cost of changing litter between broods varies greatly and depends on the cost of the litter and the labor cost of removing and replacing it. To reduce the condemnation rate, or to maintain a low level, a good sanitation program would seem economically justifiable.

I believe that houses should be cleaned between every brood. All litter, dirt, cobwebs and dust should be mechanically removed and properly disposed of. This should include all the accumulation in and around the house, the feed and boiler rooms, and the bulk storage. One can often find trouble on an untidy farm where the weeds are not cut, and especially when dead birds are not disposed of properly.

In addition to physical cleaning, the house, feed and boiler rooms and the equipment should be disinfected with a proper disinfectant. It is a lot easier to do a good clean-up job if the floor is cement and if labor saving equipment is available.

The house, brooder, and equipment should be ready for the chicks when they arrive. Brooders should have been in operation long enough so that the house is dry and warm. A guard should be in place to prevent the chicks from wandering too far from the heat. At least 4 inches of new, dry, clean, highly absorbent litter, free from molds and excessive dust, should be well distributed. Satisfactory litters include wood shavings, sawdust, peanut hulls, or processed sugar cane. Precautions should be taken to see that no one tracks disease into the clean house. Every one entering the house should be forced

to walk through a foot bath containing a proper disinfectant used in the right amount.

The brooding period is a critical one. Heat should be adequate so that proper ventilation is possible. A good poultryman knows when the chicks are comfortable. Adequate and easily accessible feed and water space are essential. Feeders and waterers should be kept clean. Waterers should be cleaned daily; the feeders, when needed.

Proper environment for the broiler requires adequate housing, brooding, feeding and watering facilities. The best chance of success is a combination of a good poultryman, good chicks, and good facilities. A good grower cannot entirely overcome an inadequate brooding system or a poorly ventilated house. Often, constant temperatures are impossible to maintain because of construction and heating facilities. Ventilation problems result when an attempt is made to maintain satisfactory temperatures. There may be no best house or brooding system. At least, it would be impossible to get an agreement on what it is at the present time. However, it is pretty well agreed that chickens should be comfortable at all times. That usually means enough heat under all conditions so that enough ventilation can be given to supply fresh air without chilling the chickens.

The broiler grower must be on the job, especially during the winter months, to anticipate rapid changes in temperature and humidity and make the necessary changes in the amount of heat and ventilation. Failure to use added heat, when needed, is one of the most frequent causes leading to high condemnations. During periods when the price of broilers is below cost or marginal, and to a less extent at other times, some increase in condemnations is due to growers discontinuing heat too soon to reduce the fuel bill. A rapid drop in temperature and an indifferent operator often combine to give the chilling shock that causes trouble.

To get well-feathered broilers, houses should be kept as cool as possible, provided the chickens are comfortable. It takes some judgment in the regulation of heat and ventilation to prevent overheating and chilling. When there are wide variations in temperature and especially when the humidity is high, it is better to keep the brooder in operation and cool the house by additional ventilation from side panels than to remove the hovers or turn them off and close up the house to try to maintain an adequate temperature. Under most conditions, ridge ventilators should be closed during the winter months and until weather becomes settled in the early spring.

There are many general management details the broiler grower should consider. Most of these will be discussed in detail in another paper.

The poultry grower will often depend upon a service man for his vaccination program. A good program should be adopted and used consistently. If vaccinating crews are used, every reasonable precuation should be taken to prevent them bringing in disease. The safe procedure is to have each man wear a clean pair of coveralls at each farm. Whether or not the cost of doing this is justified is questionable.

At all times, culls should be killed as they show up. Runts will not grow fast enough or use their feed efficiently enough to pay their way. In addition, they are always a potential source of infection.

Dead birds should be disposed of immediately. A disposal pit, adequate in size and design, should be conveniently located. On January 6, 1960, the State of Georgia made it mandatory for all poultry producers to have adequate sanitary disposal facilities for dead birds. More states should make similar rulings.

In times of trouble, an indifferent attitude on the part of the grower is really serious. He will fail to listen for respiratory symptoms. He will fail to note a drop in feed intake indicating a depressed appetite, an increase in mortality, or picking, which is the forerunner of cannibalism. If he does observe the trouble, he may fail to contact soon enough the service man, the county agent, the extension poultry-man, the pathologist or anyone else who can help him.

The good grower does all he can to get a diagnosis as soon as he can, provided conditions warrant it, and provided he has not become discouraged and disgusted by past experiences. He gets that way if he, or whoever tries to help him, travels several hundred miles to two or more diagnostic laboratories and gets conflicting reports of what the trouble is and gets no help. In cases of respiratory outbreaks, the grower should endeavor immediately to stimulate appetite with an antibiotic

feed, strive for increased feed and water intake by increasing feeder and water space, remove culls, keep down dust, and increase heat so more ventilation can be given.

Good management goes hand in hand with sound nutrition and feeding practices. These have been fully discussed in another paper.

In conclusion, management practices are an important part of any program for the reduction of condemnations. Most broiler growers know, or can easily find out, what is thought to be the best program. However, it must also be kept in mind that the best programs we have today are based primarily on field observations and that, although the findings are considered practical, the conditions have, in most cases, been uncontrolled. The observations so obtained actually apply only to a special set of changeable conditions that happen to exist at any particular time. There is a need for a great deal of research in which as many factors as possible are under control and from which basic principles can be established.

In the meantime, a considerable reduction in condemnations is possible if broiler growers will do what they know they should do to grow good broilers.

PHYSICAL FACTORS THAT CAN INFLUENCE TRANSMISSION OF POULTRY DISEASES

By William R. Hinshaw[1]

This discussion on physical factors that can influence transmission of poultry diseases will be limited to those of special importance to the broiler industry. Emphasis will be placed on those that can influence the rearing of broilers to marketable age with maximum wholesomeness. The primary aim will be to suggest problems that need answering by engineering research, rather than to attempt to enlighten the producer on current methods of prevention and control of disease. I want to state at the beginning that I am not an engineer so my remarks will be biologically biased.

We are in an era of specialization. Poultry husbandry as an art is, or has already been, replaced by many specialties and technologies. In this specialization one must not overlook the fact that we are still dealing with a complex biological entity from the egg to the finished product, whether it be a broiler or a roaster. This is especially important when research tending towards automation is considered. One cannot develop a production line operation with a live biological product by the same techniques that he can use in the manufacture of airplanes or automobiles. It is important that other scientists as well as engineers keep this biological factor in mind in planning cooperative research programs.

Dr. T. C. Byerly (2)[2] in discussing goals of engineering research on poultry housing at the American Society of Agricultural Engineers, Farmstead Engineering Conference on Confinment Housing of Livestock held in September 1960 at the University of Illinois stated that:

> "First, basic research must provide the physical and much of the physiological data on heat and gaseous exchange needed to make life of poultry in a restricted environment possible.
>
> Second, engineering research on poultry housing must provide much of the physical data and aid in determining the

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[2] Figures in parentheses refer to References at end of this article.

physiological data on heat, gaseous, and water exchanges, and on light, temperature, humidity, and air movement in optimal and also less-than-optimal but acceptable environments; and devise structure-equipment complexes that will dependably produce such environments.

Third, it must provide information which will lead to the provision of housing at lowest sustained cost in terms of cost per unit of production. A corollary is that housing must in some measure meet the convenience of operators and the social habits and health requirements of poultry."

Dr. L. A. Wilhelm (13) has suggested a new term "minimal environmental control" (MEC), and defines a broiler house having MEC as follows:

"A broiler house with MEC has a reflective roof, with paper barrier; revolving roof ventilators; hose sprinkler on the roof; fans; removable windows; slide drops; and circulating hot water heat."

These men have outlined in these statements ideas for a lot of engineering research of interest to the economic rearing of broilers under all types of environment and degrees of health. The ideal light, air movement, temperature and humidity control, types of feeding and watering equipment, and the spacing of these are examples of engineering problems that are immediately thought about. The brooding equipment, and especially the effect of types, source of heat, and methods of control become engineering problems of concern. Types of flooring and litter used on floors must be considered.

Everyone accepts the premise that one of the major research problems concerns profitable rearing of chicks to a marketable broiler age and size with maximum gain and minimum condemnation. It is a known fact that many lots of chicks have been already exposed to one or more diseases, including so-called chronic respiratory disease (CRD), when they are placed in the brooder house. For this reason the problem immediately becomes one of "convalescent rearing." I am sure that many of you will object to the use of this terminology. It is a fact, however, and an important phase of the agricultural engineers' mission in the proposed cooperative program is research on designing convalescent rearing units and equipment which will best aid in helping normal chicks to resist infection from exposure to those that are sick, and to provide conditions for rapid recovery and maximum growth of the convalescing chicks. The problems, therefore, become in many respects comparable to design of infection and isolation wards in hospitals, with special reference to diseases of children because of the age limits of the subjects under discussion.

The basic principal for economic rearing of broilers is still the use of disease-free breeding stock. The cooperative research program should be directed towards the elimination of brooder house diseases. An engineer would be the first to admit that he or his profession cannot hope to design equipment, houses, ventilation systems or total air condition systems that will make it possible to rear sick chicks as economically as disease-free chicks even under the most ideal conditions. However, if maintenance of disease-free flocks is not possible, engineering research on housing and environmental factors as they affect the convalescing chick becomes most important. The principals and techniques developed for convalescent rearing should make normal (disease-free) rearing easy.

The popular approach to a dissertation on the physical factors that can influence disease transmission is a discussion of the overworked subject of stress. The existence of disease in the brood, either because of a percentage of the chicks being infected on purchase, or because of a contaminated house and equipment, is a stress factor in itself. In either instance the brood starts its short life with a distinct handicap. The immediate problem then becomes one of making conditions ideal for the exposed chicks to eat and drink as normal as possible, to reduce to a minimum the chance of spread of the existing disease so that a reasonably sound, meaty, wholesome carcass can be produced.

No other type of livestock varies as greatly in metabolism as does the fowl. When one considers the expired air of an 8-week old broiler in relation to its metabolism and the ambient air of the brooder, it is the air at 6 to 10 inches from the floor level--not 5 to 6 feet as

in the case of man or the horse or the cow, or at 2 to 3 feet as in the case of smaller animals like sheep, swine, and dogs. It is equally necessary to consider air analysis for presence of aerosols of disease, ammonia, CO_2, CO, or other gases at this height. For example, ammonia gas has an air density at 0° C. of 0.596 according to Longhouse et. al. (8). They also state that ammonia can be detected by humans when air contains 10 to 15 parts per million (p.p.m.) and eyes begin to water at 50 p.p.m. According to them, chicken hens will stand 40 p.p.m. Scarborough (9) has shown that growing chickens exposed to 50 p.p.m. for 10 days will be affected.

It is important that the physical effects of ammonia and other gases on birds be determined and that quantities in air examined be stated in terms of distance from floor levels and especially at bird levels. Very little information seems to be available on litter decomposition, and the gases that are evolved or the effects of these on either normal, sick, or convalescing birds. Such information would be of use in evaluating housing construction, air conditioning, and brooding equipment research. Even under the best of air filtration systems, birds and man both may generate more aerosols within the house than come in from outside air. Therefore, the rate of change of air may be very important in keeping infection reduced to a minimum.

Under most circumstances it is the removal of infected air that is more important than the filtration of incoming air. Research on quantitative microbiological content of air within houses at varying levels from the floor as well as of the outside air is suggested as a means of evaluating the possible role of air in transmission of diseases in broiler plants. Particle size studies should be included. Two references related to air sampling that may prove of value are: (1) Wolf, et al. (15) on sampling microbiological aerosols; and (2) Decker et al. (3) on air filtration of microbial particles.

Brooders and methods of brooding may represent stress factors which can influence adversely the development of the brood. Again, the problem may vary with the health of the brood. Certainly, convalescing chicks require different brooding conditions than good healthy ones. Brooder management studies need to consider methods of handling the convalescing brood and the normal one as a means of salvaging as high a percentage as possible for market.

For example, Hudson (5) in a study on brooder mangement found that 92° F. (at the manufacturer's thermostat) produced slightly heavier chicks than did a temperature of 97° F. and much better chicks than those brooded at 87° F. With thermometers placed at the edge of the brooders and 4 inches from the floor, the ideal temperatures were 80° to 90° F. These data were supposedly collected on healthy chicks. If convalescent rearing is going to be required, data on ideal brooder conditions for chicks recovering from CRD and probably other diseases are needed. Drury and Baxter (4) state that usually no attempt is made to control humidity under brooders. Under certain conditions, however, humidity may be an important factor in salvaging a profitable number of birds. Quantitative examination of the air under brooders for gases may also be worthwhile in studies on the effect of brooding techniques on chicks.

Automation of feeding and watering systems could be an important cause of increased mortality and/or condemnations. I understand that many growers using automatic equipment find that occasional inspections or even walking among the birds aids in inducing them to eat and drink. In other words, a substitute for the attendant, whose mere walking through the plant inspecting the feeders as means of encouraging eating, has not been incorporated in the automatic equipment. In convalescent rearing, this personal attention can become an important factor in inducing chicks to consume enough feed. Several years ago when we were doing research work on hexamitiasis in turkey poults at the University of California, we found that frequent visits to a pen of sick poults and adding feed to the hoppers frequently encouraged them to eat and drink. This personal attention alone aided in markedly lowering mortality. I am sure the same procedure would pay dividends in many instances. It is possible that engineering research may be able to improve present automatic equipment to correct this minor but important deficiency.

Reduction in mortality as a result of treatment, or other management of sick birds may yield more broilers for market. If, however, the chicks saved have not

been held long enough to allow time for healing or for change of air sac lesions from an exudative to a residue stage, condemnations percentages may be increased. The mere physical holding of such birds for a few days may be a necessary change in management practices if it is impossible to produce disease-free stock.

Next to a sick bird or a normal carrier (shedder), man is the worst offender in mechanical transmission of disease from one area to another. This well known fact needs no elaboration, but one personal experience will serve to emphasize it. At the University of California I gave a student a class problem on mechanical transmission of coccidiosis. To accomplish this he walked through a brooder house heavily contaminated with coccidia. From the contaminated house he walked about 1 mile over dirt and gravel paths and then washed the soles of his shoes with water. These washings were mixed with feed which in turn was given to normal susceptible chicks. The result was a severe case of coccidiosis.

I am sure that engineering research will not improve the habits of man so that he will refrain from entering poultry houses, feed rooms, and other "out-of-bound" areas after coming from known disease areas. Much can be done, however, to discourage this kind of traffic by improving the construction of houses, use of self locking entrance doors, and improving storage facilities. Time will not permit detailing suggestions but they include construction of feed storage equipment outside the building with easily accessible loading openings also outside, and litter and manure disposal systems that require minimum of human traction within the pens. This hardly the place to elaborate on the need for supply of ample clothing and boots for use of personnel needing to go into houses, but it is an essential requirement.

"Disease-proofing" a house not only requires proofing against the mechanical carrier known as man or Homo sapiens but against other well known physical carriers such as rodents, other animals, wild birds, and arthropods (insects). Careful consideration of design of feed storage facilities; carcass, manure, litter, and other refuse disposal systems; drainage systems; water supplies; and even of location of the buildings in relation to each other and the general terrain will do much to aid in control of these problems.

The importance of dust as a factor in transmission of disease, either as it is developed from within a poultry house, or as it is circulated into the house from the outside, depends on the character of the dust. If disease-carrying microorganisms are prevalent in large enough quantities, if they are of critical particle size, and if the relative humidity is at a proper level, dust can be hazardous in disease spread. Dust can also cause stress as a mechanical irritant or as an allergen-carrying vehicle. In any case dust control is essential and of engineering research importance. Research on dust control involves air conditioning and ventilation problems relating to filters, amount and frequency of exchange of air. Location sites can become important both from the standpoint of the terrain and prevailing wind direction. Nearness to other houses and the location of ventilation systems in respect to other buildings can become important.

Recently in a nearby state, the State Health Department became concerned with an increased incidence in operation wound infections in one hospital. This hospital had had a previous history of gas gangrene, and on investigation it was determined that the physical location of the hospital building played the most important role in the contamination of the operating rooms, equipment, and subsequent infection through operation wounds. The soil in the neighborhood was found to be heavily contaminated with a spore-bearing bacterium responsible for the infection in surgical patients. The prevailing wind and even a local railroad passing nearby played roles. The local train was found to stir up an unusual amount of dust during dry periods, and the wind carried aerosols of the bacterium into the hospital. Such a situation could exist in a poultry plant and I am sure it does exist.

The example is given to illustrate the importance of environmental research. Engineering research on factors concerned with terrain and location can play an important part in solving some of the existing problems of disease transmission. Filtration systems, retardants for dust within the houses, and control of relative humidities, become research problems in efforts to control dust.

SUMMARY

I have attempted to limit this discussion to those physical factors which can influence transmission of diseases of importance to the broiler industry. The factors I have discussed are not new, but I hope repetition of them has been of some value and will suggest a few new ideas for research. I am sure that the combined efforts of the array of engineers, geneticists, nutritionists, physiologists, veterinary scientists, poultry husbandmen, and bio-statisticians planned for the cooperative research programs in Georgia and Mississippi will be able to solve many of the existing problems.

Engineering research aimed at improving conditions for "convalescent rearing" of broilers has been stressed. This terminology has been used to emphasize a realistic problem which faces research workers for immediate future planning. As stated several times during the past few days, it is necessary to face the fact that until biological research is successful in developing methods of eradicating the diseases causing losses in broilers the producer has the problem of raising chicks that have been exposed to disease when they are purchased. Until such a time as research makes it possible for the broiler producer to obtain disease-free chicks, engineering research as well as a share of biological research should be centered around improving facilities, equipment and rearing techniques that will aid in salvaging the highest percentage possible of broilers as marketable and wholesome. Such research should only serve as a stopgap, because the ultimate solution will be development of disease eradication procedures.

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SECTION V--ENGINEERING CONSIDERATIONS

ENGINEERING FACTORS RELATED TO POULTRY CONDEMNATION LOSSES

By Robert H. Brown[1]

There is much to challenge today's engineer. Those engaged in the profession are diligently seeking ways and means to comprehend the full significance of the challenges and to so organize their efforts that the most effective solutions will result. Engineers are concerned with the production, use, handling and conservation of materials and machines; with the control, instrumentation, quality and performance of products; and with the transformation, transportation and utilization of energy.

Some in our profession are involved in the challenge of space. This example is cited for its implications of how scientists and engineers, trained in various disciplines, work together to attain a common goal. We have gathered here to involve our minds and join our efforts in the challenge of poultry diseases and more specifically in the challenge of reducing condemnations of broiler class poultry. There are multiple points of beginning. In fact many investigations are underway. From these efforts, from prior work, from management procedures, production records, observations, hopes and guesses come many suggestions about the part engineering should play in helping to reduce condemnations.

Popularized by their reasonableness, by the large number of supporters and by the lack of data to the contrary have been these bits of advice: To reduce condemnations, it is necessary to keep the birds warm and the house dry and well ventilated, eliminate drafts, provide plenty of fresh water and feed, prevent wet litter, disinfect, insulate, and control ammonia levels. Included here could be gift-wrapped solutions to our problem but they are not in usable form. Engineering factors are involved, it is true, but the engineer must have numerical values, not adjectives, if he is to be of service.

Prior to directing attention to specifics, let us consider an engineering view of broiler production. Viewed as an entire system, it is a problem in the handling of materials, FETD, "from egg to drumstick." And the complete benefits of technology cannot be truly effective until the separate stages of production are considered in this view. Improved economy and upgrading of the product is our desire. In bringing this about the incubators, broiler house, and processing plant might end up at the same general location. Air, heat, feed, litter, water, radiations, cooling and cleaning would be handled automatically and mechanically. The handling of the birds would be mechanized almost completely. But there are many variables and design criteria involved in the design of such a system. In fact, as of today, it cannot be done for lack of basic information, and one area in which information is lacking concerns the relationship of certain parameters of environment to poultry diseases. Keeping these ideas in mind, let us now consider the production cycle.

The incubator represents the one place in the production cycle where the environment is closely controlled. Not much is desired here in the realm of hatchability, but there is a question of stress perhaps induced by fumigation or hatch time which might later prove to be a factor in causing the chicks to be more susceptible to respiratory infections. Quality control of products is a necessary and integral part of any production system and baby chicks

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should not possess differences as a result of incubator treatment. The incubator, its controls, their calibration, check and performance are of interest to the engineer.

The next step in the system involves transporting the birds to the brooder. The incubator and the brooder should be located in close proximity if an orderly and efficient flow of materials is to be obtained. Presently there appears to be much room for improvement in this operation. With the present system, engineers recognize many factors of interest; namely, the handling of the birds, involving; packing cartons, ambient air condition during transportation and prior to being placed under the brooder, elapsed time, noise, and vibrations. To produce a uniform product differences must not be introduced here which result in immediate or delayed variations in the performance of the bird. Lamoreux and Hutt (4) reported that baby chicks do not possess the body temperatures of adult chicks until they are 10 days old. Sturkie (10) found that baby chicks up to 7 days old do not shiver when chilled and have practically no resistance to chilling. Apparently this step in the production system merits attention. Do we know for certain that the birds have not been stressed unduly and unnecessarily? And if there is a stress, will it be a factor in subsequent respiratory troubles and condemnations?

The next operation is a major one, the brooding and growing out of the birds. Most of our interest is centered around this step in the overall system since it is here that time, management practices, disease, environment, and costs enter the picture with emphasis.

Many engineering principles have application in the various operations in the broiler house. We simply have not scratched the surface in bringing to this worthwhile and beckoning enterprise the bountiful fruits of technological advancements. Many structures are merely confinement areas, nothing more. Hand labor is used, at least in part, in ventilating, disinfecting, feeding, watering, handling the birds and in handling the litter. We use only a minor fraction of the cubic content of the enclosure. Our materials-handling engineering grade is F. Why? Because of a situation that grew up without consideration of these things and because of insufficient design data, basic factors, which must be supplied before we can proceed to remedy the situation.

Many of these design parameters are apparently related to condemnation losses. They are: Temperature, humidity, air composition, air movement, ventilation rate, radiations, litter, interior surfaces, and watering and feed troughs. Singularly or in combinations these factors are perhaps the key to meeting the condemnation challenge. There is much interrelationship between them and time does not permit a complete discussion of each. But it is important to consider some that are prerequisites to the design of any broiler growing system.

Temperature: Of all engineering factors, temperature is the most familiar. It can be approximated cheaply and measured expensively. It is the indicator of health and comfort most often discussed and in relation to broilers, the environmental factor with which most work has been done. Poultrymen generally agree that ambient temperatures in the range from 60° to 70°F. are best for production. The question about temperature, then, is not what is best but what deviations from the ideal shall we permit. Is it a fact that temperatures below 45°F. result in higher condemnations and poorer feed efficiency? If so, what duration of exposure is harmful to the birds? Are rapid changes more harmful than gradual ones?

Although a reduction in performance accompanies the higher temperature range (above 70°F.) it is suggested that condemnation rates are not affected as drastically by the higher temperatures as they are by lower temperatures. A possible explanation is that although stress is induced it occurs at a season of the year in which the houses are opened-up and the added ventilation rate brings about environmental changes which partly offset the influence of the stress.

Another possible temperature effect about which we must be careful is that of uniformity. The engineer must know if he is to provide a uniform temperature throughout the space occupied by the chicks, and if there is a favorable range for litter, wall and ceiling temperatures.

Humidity: Very few experiments (9, 12) have been designed to measure the effect of relative humidity either on the performance of chicks or on condemnation losses. Yet, it is frequently stated that relative humidity is one of the principle factors

involved in bird comfort. Since the birds do not sweat, respiratory evaporation is their most effective cooling media, and high humidities--especially at high temperatures--reduce the efficiency of this cooling method.

We urgently need more information on chick performances when subjected to various humidity and temperature combinations and when subjected to the 60° to 70°F. temperature range under various humidity conditions.

Dr. K. H. Kingdon has suggested that there is a range of humidity most favorable to the spread of air-borne virus. This means that there is a range which is unfavorable. The idea, in brief, is that the virus is contained in small droplets which also contain sodium chloride. The rate at which the sodium chloride crystallizes depends upon the rate of evaporation of the droplets and therefore upon the relative humidity. The concentration of salt appears to be the determining factor on whether the virus is killed, unaffected or strengthened. If the humidity is too high, no reaction takes place and the virus lives. If the humidity is too low, the salt crystallizes too rapidly and leaves the virus alive. Some place in between may be the range we desire for reducing condemnation. An engineering design excluding humidity control is simpler. If control or limitation of humidity to a specified range is desired, it can be done but at an increased cost.

Ventilation: Exchanging some of the air inside a broiler house for some of the air outside the house does several things. It removes heat and moisture, and assists in maintaining air purity. It represents one method of altering the temperature, humidity, ammonia fumes, and moisture within a space. Therefore it might be related to condemnation losses. Ventilation, like insulation, is a means to an end; it is a tool that can be accurately utilized only after the end results are chosen.

To date the predominant ventilating system in Georgia broiler houses is a manually controlled arrangement adjusted by judgement. Mechanical systems, where employed, are designed to remove heat and moisture in amounts proportional to their production by the birds. Sufficient data is available (3, 5) on heat-moisture production. But information is not available as to the best temperature, humidity, ammonia level, and litter moisture; so we may not be using the ventilation tool to the best advantage.

Data are lacking and condemnations may be unknowingly affected by other aspects of ventilation; namely, ventilation rates, ventilation methods, and air velocity For example, consider the natural ventilation rates in summer months when houses are opened up. The rates are much greater than they are in winter with the houses closed. Condemnations are reported to be less in the summer. Why? Perhaps the rate of ventilation is a contributor if not the cause. Since temperature, humidity, air velocity, ammonia content, and air motion also change when the house is open, it is impossible to identify any single factor as the cause. But this situation does add emphasis and motivation to the desire for more experimentation with the rate and method of air change.

Air Movement: Moving air through a broiler house is, of course, related to ventilation. But air can be circulated without exchange, and various types of air motion can be used to achieve a desired ventilation rate. The air velocity, both its magnitude and its direction past the chicks, may be related to stress and to the spread of disease. Information of this type is needed both for ventilation system design and for a determination of the value of fans in the summertime (2).

Litter: Since the litter must be handled, serves as the "floor" of a structure, and can store and release moisture, it attracts interest in any future engineering design. It is also a definite suspect as a contributor to condemnation losses. Litter, because of its position in the house, its physical properties and especially if it has a high-moisture content, appears to be a natural for spreading disease. We need to know more about it. Damp litter is known to increase the ammonia content of the air and apparently causes added stress on the birds.

It appears that the merits and demerits of litter should be carefully evaluated with a view towards the desirability of substituting some other type of "floor" in our broiler houses.

Interior Surfaces: Interior surfaces which are hard to clean, which tend to absorb moisture, or to which dust adheres easily might be related to the spread of disease. Certainly if the surface presents

sanitation problems, it is likely to contribute to condemnation losses.

Waterers: It seems reasonable to assume that disease is spread by chicks drinking from the same waterer. Perhaps radiations in the ultraviolet range or ultrasonic treatment of the water would be beneficial. In addition the water could be recirculated, treated or changed automatically and periodically, or made available in much larger quantities.

Transporting Coops: The coops in which the chicks are placed for transporting to the processing plant represent still another engineering interest which may be related to the spread of disease. The coops also represent a breakdown in efficient handling of materials. Some experimentation has been done on ways of cleaning the coops (11). Apparently the possibility of spreading disease from one farm to the next will be less likely if the coops are completely cleaned before they are brought to a farm.

Other Factors: These remarks have been confined to existing situations and conditions. Engineers are also interested in utilizing all areas of technology to assist in the condemnation challenge. Such assistance might be indirectly utilized such as the possibilities in the realm of new and better instrumentation. It might occur in the form of new applications such as radiated energy of various wavelengths, polarized atmospheres, and ionized air.

It is beyond the scope and time limit of this paper to discuss these added factors. They are perhaps outside the realm of the subject matter envisioned when the topic was assigned. But they could easily hold the key to real assistance in disease control, to early detection of disease, to a more rapid evaluation of stress, and to a more economical solution of our problem.

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THE ROLE OF AIR IN THE TRANSMISSION OF DISEASE

By Robert J. M. Horton and A. Nelson Dingle[1]

In considering the role of air in the transmission of disease, it is first important to recognize that air forms part of the environment for everything on the earth. Further, it is important to remember that air is a fluid capable of all the modes of behavior which fluids exhibit. It is constantly undergoing change. It is capable of serving as a vehicle for various contaminants; it has erosive action; and can transfer heat, momentum and other properties by convective action. In addition the air has water vapor as one of its important constituents.

In disease transmission we are not only concerned with the character and behavior of the air, but with certain contaminants that it contains. Of particular interest are microorganisms, especially pathogenic ones, which originate from two sources. The major source consists of infected individuals who are constantly shedding or expelling these organisms into their environment from various surfaces, especially from the respiratory tract. A lesser and more recently discovered source is soil and decaying organic matter. There are apparently a number of spore-forming organisms that are able under certain circumstances to grow as parasites in animals, e.g. Histoplasma capsulatum and Coccidioides imitis. Other air contaminants of different types, such as pollens, dusts, smokes, fumes, and gasses find their way into both indoor and outdoor air from various sources in nature and the works of man.

In this paper we wish to consider the role of air: (1) as an environment of disease organisms, of vectors of such organisms, and of the receiving host of these parasites; (2) as a vehicle capable of transporting various contaminants from place to place with varying effectiveness; (3) as a fluid subject to processing by both natural and artificial forces that are capable of rendering the air completely uninhabitable, or of improving it for animal habitation.

AIR AS ENVIRONMENT

Although air consists mainly of nitrogen and oxygen, much of the interest in air as an environment centers about a number of substances present in smaller and more variable amounts, and in some of its physical properties. At present there is great interest in complex contaminations such as smog. The effects of smog on living things are frequently dramatic and devastating, and probably also subtle as well. Whether or not contaminants of this type affect microorganisms or alter the susceptibility of animals to infection is not yet known.

MICROORGANISMS

Air contains no nutritive elements for microorganisms. It must be, therefore, a transitional environment in which they survive more or less well. With the exception of skin bacteria and spores, most microorganisms enter the environment in a moist state. They frequently come from a warm environment also. The most important characteristics of the air which relate to their survival appear to be temperature, moisture, and ultraviolet radiation. They are particularly sensitive to the latter, with the possible exception of spores which have not been well studied in this respect. Low temperatures are in general more favorable for survival than high, since they reduce the metabolic rate that allows longer viability in the absence of food, and also lower the rate of loss of moisture by reducing the saturation

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vapor pressure of the air. Most organisms are quite sensitive to high temperatures. The effect of moisture is variable and is in some instances closely related to temperature.

Many bacteria and viruses are quickly killed by drying, whereas others will survive better in dry environments. For example, it has been known for many years that smallpox and vaccinia viruses can be kept for long periods in the dry state, but die quite rapidly when wet unless they are refrigerated. The seasonal fluctuation of smallpox apparently depends on this characteristic since it is most frequent when the vapor pressure is lowest (12).[2] It may seem strange to emphasize the importance of moisture in the survival of microorganisms, since all, or nearly all of them are now commonly stored in the lyophylized or dry state. This process is, however, a complex and artificial one. The relationship between lyophylization and normal survival in air is discussed in the very interesting chamber experiments of Dunklin and Puck (7) and of Lester (9). This work merits close study by anyone interested in the problem of survival of microorganisms in air and dust. The indications of great sensitivity of these organisms to unfavorable influences at relative humidities near 50 percent appear to offer possibilities for the development of control measures.

VECTORS

Many parasitic organisms are transmitted indirectly by arthropod vectors. The relations of the distribution, breeding, survival, and behavior of these vectors to meteorological phenomena are well known and adequately documented. Less obvious and less carefully studied is the effect of atmospheric variables on the microorganism in the vector. Bates and Roca-Garcia (3) showed that the infection rate and development time of yellow-fever virus in Hemogoggus mosquitoes were affected by temperature, both in general being enhanced by higher temperatures with suitable fluctuation. They made no studies on the effect of humidity. Basu and Rao (2) found that both high temperature and high humidity were needed for full development of filaria larvae in Culex mosquitoes; when either temperature or humidity was reduced the larvae, though present, failed to mature and the mosquitoes remained non-infectious.

ANIMAL HOSTS

Similar to arthropod vectors, the effects of meteorological variables and air contaminants on man and other animals have been studied extensively (13, 14). The majority of these investigations have explored physiologic tolerance of, and adaptation to, extremes of variation. Some work has also been done within more normal limits of change. None of the information so derived appears to bear directly on the topic under consideration. The known reactions to changes in the atmosphere seem more likely to affect the course of established disease, rather than influence the reception of infection.

With the exception of wounds, it is apparent that the principal receptor surfaces exposed to air are the conjunctivae, the mouth and throat, and the respiratory tract. We are not aware of any studies that have demonstrated effects of atmospheric variables on these surfaces as receptors of infection, although several suggestive physiologic observations have been made (1, 8, 11, 16).

It is commonly thought that natural resistance or susceptibility to infection (other than that due to presence or absence of antibody) depends on complex characteristics, rather than on a single trait. One should remember the famous experiments of Louis Pasteur in which he showed that the normally resistant hen could be infected with anthrax by lowering its body temperature a few degrees (17). Here resistance apparently depended on a single factor, unless one wishes to add the proverbial irritation of wet hens. Perhaps the experiment should be repeated with an additional tranquilized control. At any rate it seems possible that simple meteorological alterations of modest magnitude could well affect susceptibility or resistance to infection with microorganisms.

[2] Figures in parentheses refer to Literature Cited at end of this article.

AIR AS A VEHICLE

The vehicular role of air begins with contamination. This may be due to the suspension of dusts by air turbulence or by human and animal activities. Such dusts may contain microorganisms, which are usually but not necessarily attached to other material. Other sources of contamination are the animal respiratory tract and mouth which are constantly expelling droplets into the surrounding atmosphere. Many of these droplets contain living microorganisms. The larger droplets travel for a few feet at most and then settle out rapidly where they dry and leave dust residues. If a receptor person or animal is nearby, these droplets may be inhaled directly and establish transfer of infection. This direct droplet transfer is sometimes spoken of as a form of contact transmission, rather than as air transmission. The more numerous smaller droplets that are expelled evaporate within a small fraction of a second and leave droplet nuclei, sometimes containing microorganisms, which are so small that they remain suspended in the air for many hours (18).

Once the vehicular function of the air is activated one must consider the interaction of several different effects: (1) The effect of air as an environment, which we have already discussed; (2) dilution of the contamination by diffusion; (3) transportation by means of air currents and winds; (4) deposition in terms of fallout and impaction.

Although outdoor air forms an efficient vehicle for some plant diseases, such as wheat stem rust which can be spread over considerable distances, it has rarely been shown to be a vehicle for animal infections. Where it is known to occur, the transmission is associated with dust, and has been limited to short distances, with the possible exception of violent storms (10). The only non-spore-forming animal pathogen known to be transmitted by outdoor dusts is the rickettsia of Q fever. This organism is thrown into the air by the movements of infected animals, and appears to infect people who are, at the most, a few blocks distant (4). Recent investigations of foci of histoplasmosis indicate a similar pattern, but further investigation is needed here. This may be due to dilution, to ultraviolet inactivation, to rapid fallout of organisms attached to rather large particles, or to some combination of these.

Indoor transmission of animal pathogens through the air is, on the other hand, a common phenomenon, especially in respiratory infections. Within buildings there are generally more sources of air contamination in the form of infected individuals in a given space than will occur outdoors. The burden of contamination in the air is therefore apt to be much greater. The dilution process is much less efficient indoors. Fallout is apt to be greater due to lesser degrees of turbulence. The lack of the scouring and suspending action of wind is more than made up for by a multitude of dust raising activities.

All of these phenomena are enhanced as one moves farther from the equator; more people and animals are confined for longer periods to smaller and more tightly sealed quarters. While some of these problems may be alleviated by artificial ventilation, this can increase the spread of infection rather than reduce it if improperly carried out. The relative importance of these several factors in any given disease must be determined, and may vary under different circumstances as well as with different organisms. Desirable information here would be quantitative data on dosage of microorganisms to the air, their fate under various conditions and changes, and the effects of different quantities on the receiving host. Data of this kind are available for toxic substances in the field of industrial hygiene, and have proven very useful.

PROCESSING OF AIR

Among the types of processing to which air is subject are heating and cooling, wetting and drying, irradiation, and filtration. All of these occur naturally, and all can be carried out selectively by artificial means as well. Outdoor air is subject to continuous fluctuations of considerable magnitude in all of the above categories during the day-night alternation. In the larger scale alterations represented by the generation and modification of air masses extreme fluctuations are encountered. Contaminants in the air are necessarily exposed to extremes of

irradiation, moistening, drying, and temperature. Decontamination is quite complete. However, it may be that time is the most important purifying agency affecting microorganisms in the air streams.

In the process of forming clouds and rain, particulate air contaminants tend to migrate to small growing droplets. Some of these droplets enlarge sufficiently to fall as rain, and in their falling collect and wash out additional contaminants. Other droplets evaporate leaving nuclei for later condensation and cloud or rain formation. Thus contaminants are frequently subject to extreme alternations of wetting and drying prior to eventual removal from the atmosphere.

Indoor processing of air is limited usually to temperature alterations conducive to human comfort. Thus the air in buildings is prevented in so far as is practical from undergoing the temperature alterations to which outdoor air is subject. Air motion is also kept at a low level since rapid air flow produces discomfort. Awareness of change in humidity of the air in man is much less acute than sensitivity to motion and temperature. Only extremes are annoying. Therefore there is much less attention given to this aspect of air processing. There seems to be a generally accepted concept that comfortable animals are healthy animals, and that moderate unvarying temperature is the key to comfort. It appears to be quite comfortable for many pathogenic microorganisms too. Simpson has recently reviewed this problem (15), illustrating the changes produced by winter heating, and questioning the desirability of producing warmth accompanied by very low humidity. Study of the health aspects, as distinct from the comfort aspects, of optimal levels and variations in temperature and humidity has been very limited.

Filtration and washing of indoor air is used mainly in large buildings, principally for the removal of larger particles and odors. These processes depend on inertia of particles for their effectiveness, and hence are relatively inefficient for the removal of very small particles such as microorganisms. Electrostatic or thermal precipitation are more efficient for removing particles of smaller size, but their effect on microorganisms has not been extensively studied. Procedures for the suppression of dusts in order to limit the spread of infection have been a rather frequent subject of experiment. The conflicting results of these studies recently have been reviewed by Cruickshank (5).

In recent years a number of experiments in the processing of indoor air with ultraviolet light have been carried out, particularly in schools (6). Perhaps the most important result of these studies has been to demonstrate that air transmission is an important factor in some diseases and not in others. In addition they have shown that diseases that are airborne indoors are also transmitted by other more intimate means, i.e. air transmission is not their only means of spread. In spite of the expense and work involved such processing has been felt to be worth while in selected circumstances. It should be pointed out that these experiments (and similarly directed ones with glycol and other vapors) have been very limited up to the present time. Another form of radiation, that of visible light, is commonly applied to hens to increase egg laying. While it may seem that this could have little bearing on the problem under discussion, gestation is known to increase the excretion of several disease organisms, and may in addition alter receptivity to infection.

CONCLUSION

In conclusion we would like to point out the following:

1. The role of air in the transmission of disease has been inadequately studied. This is true of its roles as an environment of microorganisms, vectors, and receiving hosts, and as a vehicle for the transmission of microorganisms.

2. The processing of indoor air has been governed too much by considerations of human comfort and insufficiently studied for its pertinence in the promotion and maintenance of health, and the reduction of disease transmission.

3. The fragmentary information available indicates that further studies would be of great interest, and would probably improve our ability to control the spread of some infections. This appears to be particularly true for the study of the effects of levels of, and variations in atmospheric moisture.

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BIOLOGICAL AIR CLEANING

By Herbert M. Decker[1]

I have been asked to discuss the subject of Biological Air Cleaning. Information derived from this discussion may well serve to orient your thinking in connection with the design of your two new research laboratories that are planned in the South. Discussions with various veterinarians in the past few months have indicated that little consideration has been given to air cleaning with the exception that some of the newest poultry houses are air conditioned.

Prior to making suggestions as to what might be done in the poultry industry, let me first review what exists for the protection of personnel against airborne microorganisms. In general, most air cleaning equipment used to remove dust, mist, or fumes from air will also remove some bacteria. There are available many types of air cleaning equipment, such as filters, electrostatic precipitators, and air washers. In addition, there is ultraviolet light and air incineration.

Over a period of years, we have engaged in an evaluation program of various types of air cleaning devices. Results of this evaluation program are shown below. The efficiency range of air cleaning devices for removing biological particles is in the 1 to 5 micron range. However, if the bacteria are associated with dust particles to give a particle size greater than 5 microns, the efficiency will be higher than indicated.

Efficiency Range of Devices for Removal of Biological Particles (1 to 5 microns) From Air

Cleaning device	Bacterial removal to be expected
	Percent
Absolute filters	99.99+
High efficiency filters	90 to 99
Medium efficiency filters	60 to 90
Electrostatic precipitators	80 to 90
Air washers and scrubbers	20 to 80
Roughing filters: Fibrous, metallic, oiled, and screen type	10 to 60

If one is interested in the cleanest possible air, then the type filter designated commercially as an absolute filter should be the one of choice (figure 1). The absolute filter was developed by the U.S. Army Chemical Corps for use in gas masks and in building filtration systems for the removal of bacteria. These original filters unfortunately were not fireproof; however, as a result of recent technological developments, it is now possible to obtain commercially absolute filters that are fireproof, and ones that can be used at temperatures up to 1,000° F. Some of the materials used at present in absolute filters are cellulose, asbestos paper, and compressed glass fibers. Although these filters are excellent for the removal of all particles down to at least 1 micron in size, it is certainly uneconomical to use them alone to remove large quantities of dust and other particles above 5 micron size. For these larger particles, a pre-filter is suggested ahead of the absolute filter.

However, too frequently we lose sight of the fact that while on one hand we attempt to remove all organisms by filtration, the people themselves continue to generate aerosols. Generally speaking, people or fowl generate more aerosols themselves than are contained in the incoming air. Certainly, under most circumstances the outside air does not contain very high concentrations of bacteria, and the use of air-cleaning devices that are 90 to 99 percent efficient will provide a very clean environment.

Filters with an efficiency of 90 to 99 percent for the removal of all particles 1 to 5 microns in diameter are classified as high efficiency filters (figure 2).

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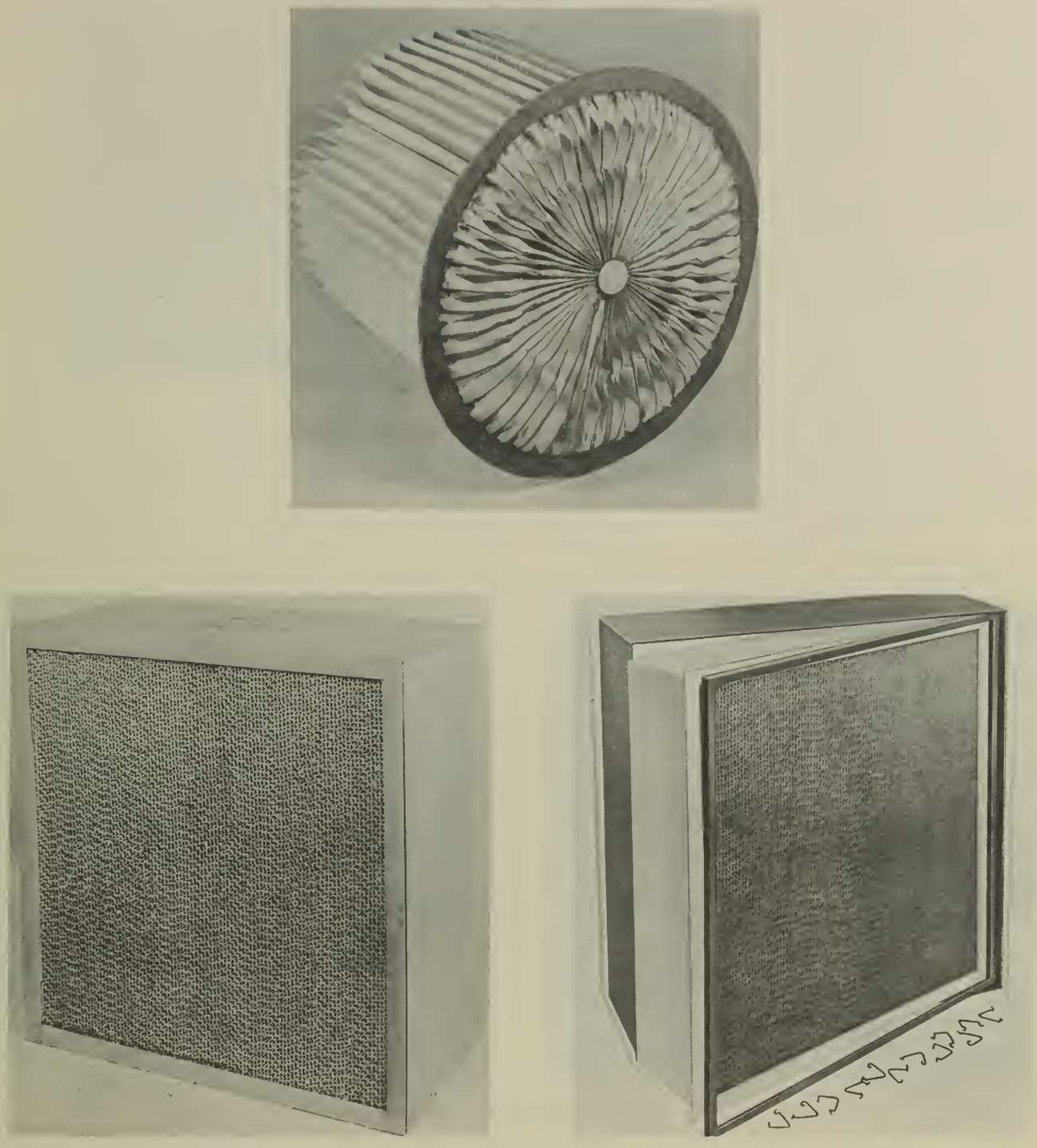


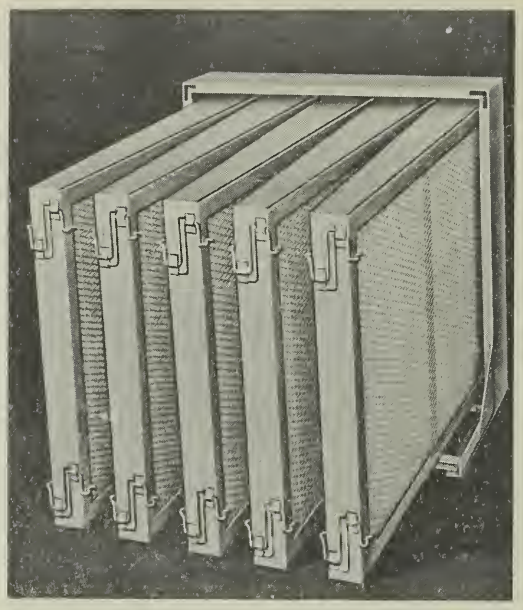
Figure 1.--Absolute filters.

The filter media are chiefly glass fibers, good grade filter paper, and asbestos fibers; the diameter of the filter media ranges from 1 to 5 microns.

There are many situations where it is necessary to remove only the majority of the biological particles present in the atmosphere. Under these conditions, such cleaning devices as medium efficiency filters or electrostatic precipitators have been adequate.

We have defined medium efficiency cleaning devices as those that remove from the air 60 to 90 percent of the bacteria and other particles in the 1 to 5 micron diameter range. The filter material is usually compressed glass fibers or a good grade of paper fiber (figure 3).

Electrostatic precipitation is a widely used method for reducing pollution of air caused by smoke and dust. Although



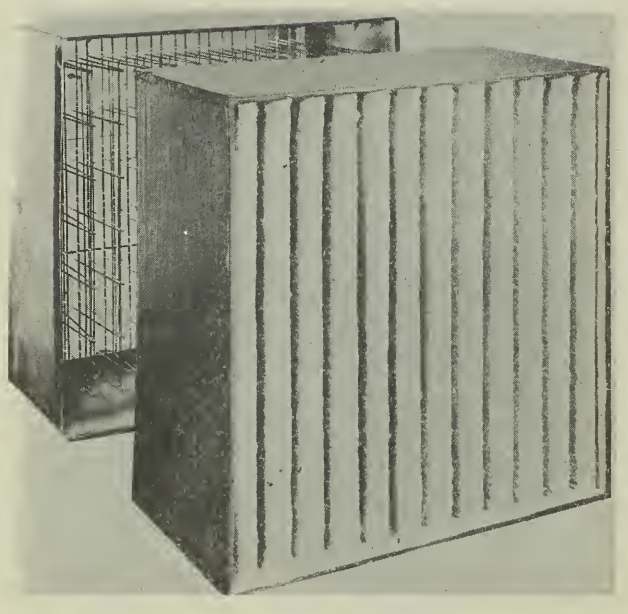


Figure 2.--High efficiency filters.

these units can remove a high percentage of bacteria and dust from air, they may not be as satisfactory as filters where a constant supply of clean air is required. In case of power failure it would be possible for the contaminated air to pass through the devices, a leakage that in some instances could not be tolerated. Electrostatic precipitators that receive maximum maintenance have been shown in laboratory tests to remove or destroy approximately 90 percent of the micro-organisms. However, tests of some units under normal operating conditions have shown much lower efficiencies. Without maximum maintenance of electrostatic precipitators, a false sense of security may be obtained.

Another method of air cleaning is by air washing. Air washing is used chiefly for removing dust and other particles from air; however, it has been used in some instances for removing bacteria. Spray towers, zigzag baffles, and metal screens constitute some of these types. The most efficient air washers are those in which the suspended matter is impinged on a wet surface and then washed off and

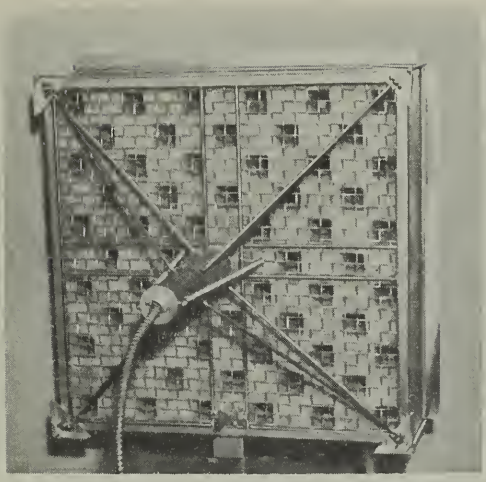


Figure 3.--Example of a medium efficiency filter.

removed. Air washers tested have been found to remove only 20 to 80 percent of the bacteria in the 1 to 5 micron range. In some instances where the wash water is recirculated, the actual bacterial count of the air increases because of re-aerosolization of the collected bacteria that accumulated in the water.

Another air washer is the type that uses a hygroscopic solution, usually lithium chloride, for control of humidity. These washers usually contain numerous rows of multi-fin coils for temperature control of the absorbent and have been found to remove approximately 40 to 80 percent of particles 1 to 5 microns in size and slightly higher efficiencies with larger particles. These washers are an improvement over the standard washers using water since there is no apparent re-aerosolization of accumulated bacteria.

Roughing filters are commonly used when large amounts of contamination and debris are in the air (figure 4). They will remove the bulk of large airborne particles, also 10 to 60 percent of the bacterial and other particles of a similar size (1-5 micron range). However, most remove less than 50 percent of 1-5 micron particles. Roughing type filters and possibly medium efficiency filters may also be used as prefilters for higher efficiency and absolute filters to remove the "sticks and stones" and to reduce "loading" of the more expensive filters, which in turn reduces total operational costs.

Two types of roughing filters in general use are the viscous-coated and the dry. Viscous filters are composed of materials, such as loosely packed fibers of animal hair, hemp, glass wool, synthetic or woven metal screens. The fibers are frequently coated with an adhesive substance, usually an oil, which aids in retaining the trapped particles. In some instances these filters are constructed for indefinite use and can be cleaned and re-oiled when the filters become loaded. Another viscous filter is the metal screen filter that is automatically cleaned when the belt of the metal screen panel moves perpendicularly to the airstream and passes through an oil or water bath at the bottom of the filter unit where the panels are cleaned and rewetted. The dust collects as sludge in the bath.

The dry type of roughing filter is composed of loosely packed glass fibers, cotton batting, paper, or other materials, and, in general, offers more resistance to the passage of air. However, they cannot be recleaned and must be discarded when the resistance to airflow becomes excessive.

For more specific information as to individual types of filter performance I refer you to the draft of a monograph entitled, "Filtration of Microbial Particles From Air" that has recently been completed and submitted to the Government Printing Office for publication. A few copies of the manuscript are on file with the Agricultural Engineering Research Division, ARS, USDA, at Beltsville, Md.

Before I conclude this very brief presentation, I would like to mention that in addition to being concerned with the relative efficiencies of air cleaning devices, we must not forget that to maintain an area free of biological contamination, we must keep it pressurized; and we must install filters correctly. In special circumstances we may find it necessary to decontaminate the filter prior to replacing it. Furthermore, we should know something about the ability of the selected filter to remove microorganisms. All these factors are covered in the monograph.

At a recent seminar on Environmental Aspects of Institutional Infections held at the Public Health Services, Communicable Disease Center Laboratory, it was concluded that as far as air cleaning was concerned, filtration is the choice method for the removal of biological particles from air, since it is the most efficient and simplest method available.

I have not covered the use of ultraviolet light or heat for the destruction of microorganisms. Numerous UV studies have been conducted, and some are

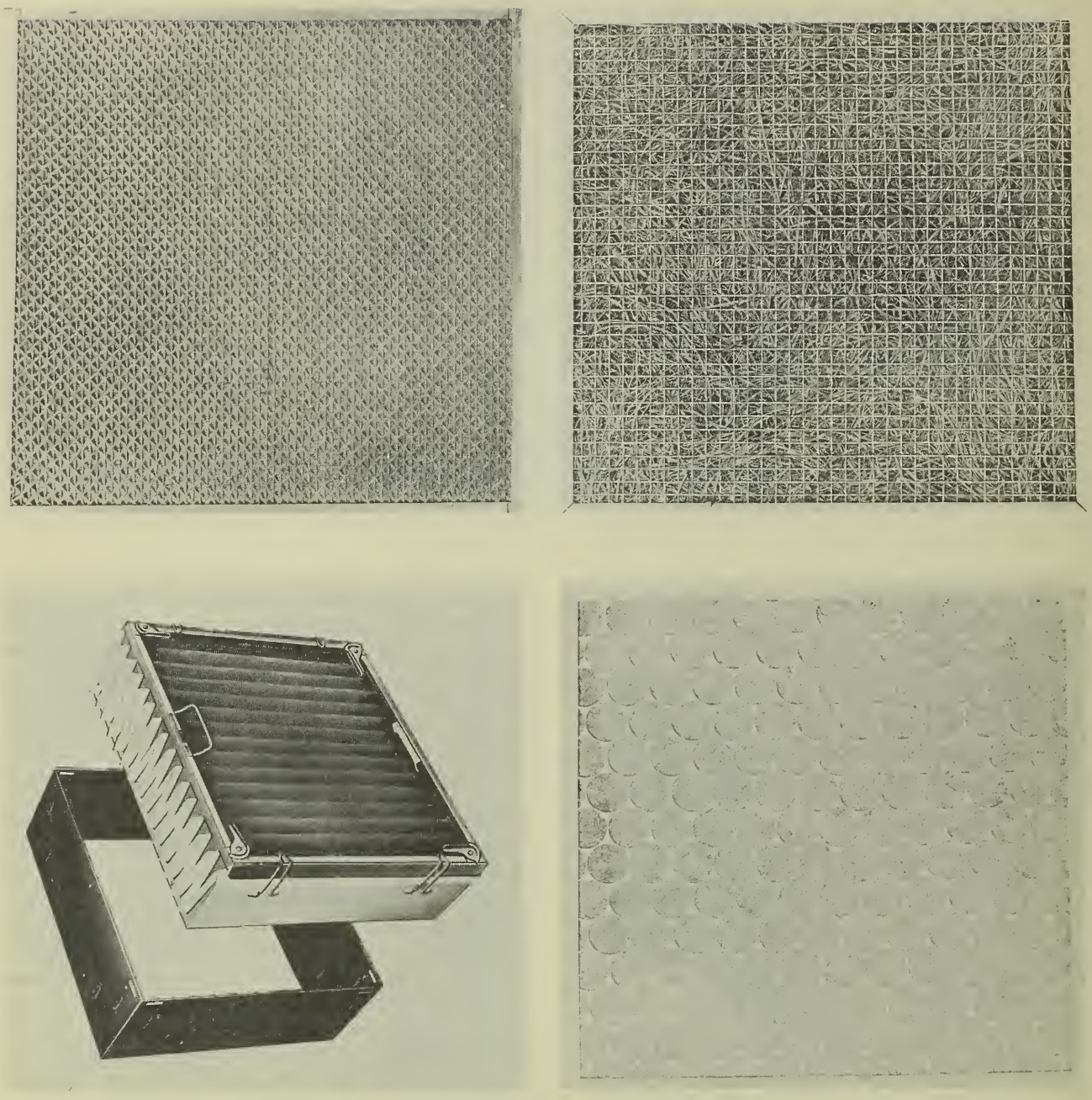


Figure 4.--Roughing filters.

presently in progress. These studies are designed to show reduction of bacteria with this method. However, this method has the distinct disadvantage that the lamps must be cleaned and tested frequently. Furthermore, UV has no capability of penetration, and therefore organisms protected by dust may not be killed. Certainly, the amount of dust and dirt found in poultry houses eliminates consideration of this mode of air decontamination.

Air incineration is a very expensive method for destruction of bacteria. The only place for this method of biological destruction is in research institutions where high concentrations of bacterial aerosols may be purposely generated for experimental reasons.

The existence of air cleaning equipment has been brought to your attention. The installation of an efficient biological air cleaning system to serve certain areas of research installations, does not, in itself,

necessarily ensure freedom from biological contamination. To maintain the atmosphere of one or more rooms at a low level of bacterial contamination, it is necessary to establish a system of differential pressurization within the building. Clean air flowing into a room, for example, a surgery, must be utilized for the purpose intended--namely to provide to the greatest extent possible a germ-free atmosphere for patients. Use of a pressurized air system permits a minimum interchange of air from areas such as corridors and work rooms, or other parts of the hospital where the concentration of bacteria will be higher than that ordinarly found in operating rooms. The pressure differential between the surgery rooms and the hallway adjoining it should be from 0.2 to 0.6 inch (water column) with the operating room having the higher pressure. To facilitate such a balance the use of cubicle or air lock shown in figure 5 is recommended. If one is entering or leaving the pressured air, the one door should be quickly closed before opening the second door.

Now let us assume that one is working in a bacteriology laboratory handling pathogenic microorganisms. In such a situation, it is necessary that the laboratory be under a reduced air pressure, so that the laboratory air that may contain pathogens (figure 6) is drawn through an efficient exhaust cleaning or incineration system, and not permitted to get into the hallways or areas where personnel can come in contact with the organisms. If there are several rooms in which pathogenic bacteria are handled, then increasing degrees of reduced pressures should be provided, the more hazardous rooms having greater reduced pressure in relation to the less hazardous areas. In this manner the direction of airflow will always be in the direction of the more dangerous area.

Summarizing what has been said--if the incoming air must be kept entirely bacteria-free, then a roughing filter followed by an absolute filter is the system of choice. Normally, however, cleaning the air to this extent is not necessary and a lower efficiency filter is adequate. High efficiency, medium efficiency, or even good roughing filters may be used where lower standards are satisfactory. Ultraviolet light and air incineration are not applicable for the problems under consideration.

Sufficient evidence has been presented to demonstrate that an area can be kept free of airborne contamination. However,

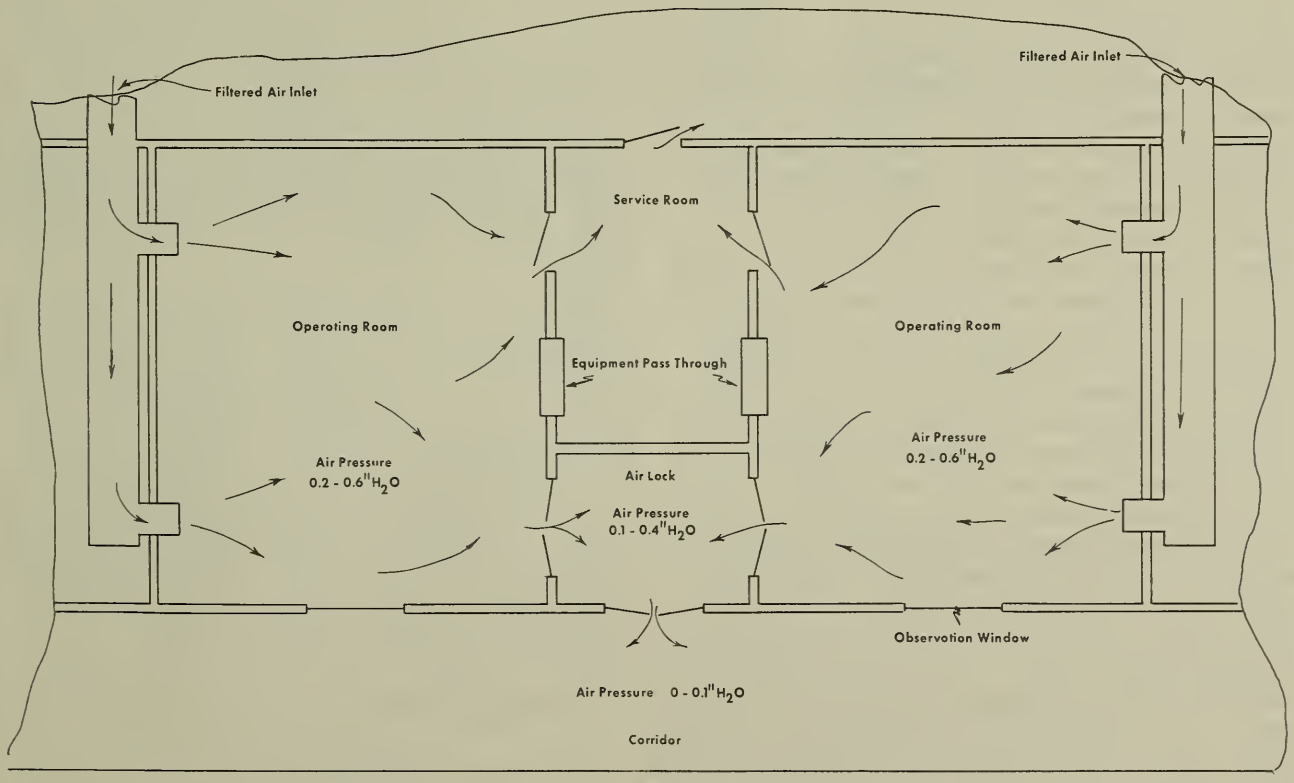


Figure 5.--Air lock to reduce infiltration of contamination.

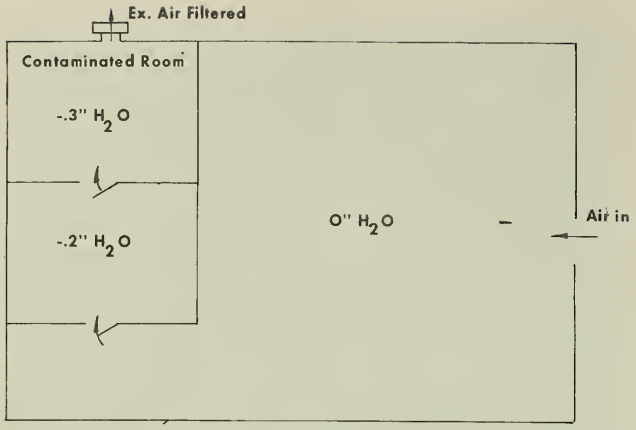


Figure 6.--Pressurized system for bacteriological laboratory.

I place before you the question--does the need justify the expense? I would believe that in those areas of poultry houses where there are forced ventilation systems and there are 10 or more complete air changes per hour, with no air being recirculated, it would not be necessary to filter air if the exhaust air ducts are not located near the inlet ducts. However, should the system utilize partly recirculated air, then you must decide if air should be filtered. Will the incidence of respiratory diseases, such as infectious bronchitis, infectious larynogotracheitis, or Newcastle in the air sac infection be lowered? In attempting to reduce the incidence of these diseases, how are you going to detect and separate within a relatively short time the diseased poultry from the healthy. This contact period may break the entire line of defense which you may be attempting to establish.

THE IMPORTANCE OF THERMAL ENVIRONMENTS IN POULTRY HOUSING

By Robert G. Yeck and Hajime Ota[1]

The words "environments in poultry housing" refer to the aggregate of all external conditions (to the birds) affecting life processes of poultry. The word "thermal" is intended to limit consideration to those external conditions involved in the exchange of heat.

The proper balance of heat input and heat output is necessary for homeotherms. Abnormal rectal temperatures generally indicate the failure of a chicken to maintain the necessary balance between input and output of heat. Loss of production, lowered resistance to disease, and eventually death occur as extremely high (or low) temperatures occur (4, 5, 6, 7, 14). This paper considers factors involved with heat exchange and some of the reported effects of these environments.

HEAT INPUT

Heat input is derived from feed and external sources. The heat derived from feed is a product of the pounds of feed and some heat conversion factor.

With fully feathered birds, external sources of heat input (such as solar radiation) may impair heat dissipation or become the final heat increment that brings on prostration when ambient air temperatures are high. With small chicks the homeothermic mechanisms are not well developed, and the insulation of the down is probably less than is obtained when chickens are fully feathered. Consequently, external sources of heat generally are necessary for chicks.

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HEAT OUTPUT

Chickens are limited in their ability to change their heat gain (input) without interfering with their productive effort. Therefore, methods of altering heat output become highly important.

Thermodynamically, we classify heat losses into two general categories, sensible and insensible. Insensible heat losses refer to losses through evaporative processes such as the evaporation of water from the outer body surfaces (skin and feathers) and from the inner body air "passages". It requires a vapor pressure gradient between the body surfaces and air. Sensible heat losses are the sum of all other heat losses and require a temperature gradient between body surfaces and the air and/or surfaces surrounding the bird.

The importance of evaporative cooling is evidenced by the fact that about 1033 Btu of heat are required to evaporate one pound of water at 106° F. The rate of evaporation is dependent on the vapor pressure gradient between the chicken and the air surrounding it, the area of body surfaces from which evaporation is taking place, and the rate of air flow over those surfaces. These factors are difficult to evaluate in any living organism and any attemps to evaluate them have required many assumptions.

Sensible heat transfer occurs through the conduction, convection, and radiation of heat. The tendency for chickens to huddle in cold weather is evidence of an attempt to minimize sensible heat loss. Conduction requires direct contact with the heat sink. It generally occurs only through pads on the foot but, of course, could occur should the birds be nesting or lying on the ground. It is a function of the conductivity of the contact surfaces, their temperature difference, and their ability to transfer heat from (or to) the point of contact. Although conduction is not regarded as an important avenue of heat transfer, it bears some consideration from the standpoint of selecting materials on which birds stand or lay. Wallowing in the litter occurs upon exposure to high temperatures and may be an attempt to increase heat loss by conduction. The role of conductive heat transfer could be an intriguing area of investigation in poultry environmental studies.

Heat transfer by convection is an important consideration of heat exchange among mature birds. It is the process wherein heat is removed by air as it moves past a surface. In poultry it is a function of the velocity and heat pick-up capability (enthalpy) of the air, the area and heat transfer characteristics of the body surfaces, and the temperature difference between air and the body surfaces. This is the mode of heat transfer involved when considering the effect of wind or fanning.

Radiant heat transfer occurs between surfaces that are separated by an air space. It is a function of the heat absorption characteristics of the air space, of the shape, position, and size relationship between the two surfaces, of the absorption and emission characteristics of the two surfaces, of the difference between the fourth power of each of the two surface temperatures, of the area of one of the heat radiating surfaces, and of the Stefan-Boltzman radiation constant. Here again major unknowns bar theoretical calculations. The emission and absorption characteristics of feathers and down and the shape factors for poultry are unknown.

Theoretical calculations of sensible and insensible heat losses by the various avenues of heat transfer can only be made through the use of assumed values. Our present state of knowledge of heat loss has been attained through empirical evaluations made possible by calorimetric devices or through evaluations of feed energy. Heat losses have been partitioned between sensible and insensible losses. The partitioning of sensible heat losses will require elaborate instrumentation now coming into use such as the 4-pi calorimeter (11, 12). Feed input energy has been partitioned according to its utilization by chickens (3, 13).

Figure 1 shows evaporative heat losses of three breeds (White Leghorn, Rhode Island Red, and New Hampshire-Cornish crossbreds) of hens at various temperatures. These data were obtained with groups of ten birds of the same breed (one breed at a time) in each of two calorimeters at Beltsville, Md. (10). The evaporative cooling ability increased with increasing ambient air temperature. This indicates that the bird had some degree of control over this method of heat dissipation. The day evaporative heat losses were definitely higher than the night losses. Apparently there are definite differences in the losses of the various

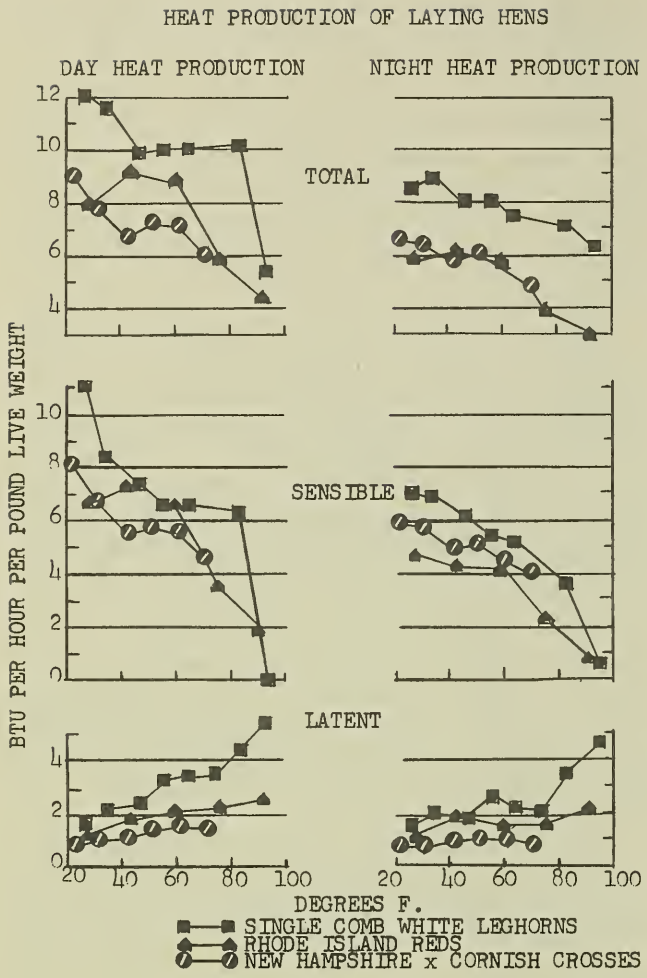

Figure 1.--Heat production of caged layers from data of Ota and McNally (10).

breeds. The higher evaporization rates of Leghorns correlate with greater water consumption by this breed (2, 10) than by other breeds.

Figure 1 also shows the total heat losses at various temperatures. The difference between total and insensible or evaporative losses is shown as sensible heat. The general trend of sensible heat loss was to decline with increasing temperature whereas that for insensible (latent) loss was to increase with increasing temperature. At 80° more than 30 percent of the total heat dissipation was by evaporative means.

An interesting aspect of the total heat loss curves was the decrease shown with increased air temperatures. For homeothermy, heat input must balance heat output. Since the bulk of heat input comes from feed, it would seem that shelters that provide a warm (perhaps as high as 75° F.) thermal environment could be justified from feed savings alone. However, the saving in feed cost must be compared to the additional shelter cost. About one-tenth of a pound of extra feed per day per 3-pound bird would very likely offset an extra 2 to 4 Btu/hr/lb. live weight heat output that might otherwise be lost in a poor shelter. The tendency for day total heat losses to be constant between 45 and 60 may reflect the ability of the heans to physiologically alter their heat transfer characteristics.

Figure 2 compares the feed requirement per pound of gain at 85° F. with that at 65° F. (9). In support of the hypothesis that greater economy of gain would be obtained at high temperatures, one might desire to see the 85° values greater than the 65° values. However, the difference, if any, favors the lower temperature. It is possible that the 85° environment was distressing enough to the chickens to cause some loss of production efficiency.

A review of literature reveals that elevated rectal temperatures have been reported at temperatures as low as 80° for some chickens (15). Figure 3 shows widely quoted rectal temperature data of Yeates, Lee and Hines, (15). Results of

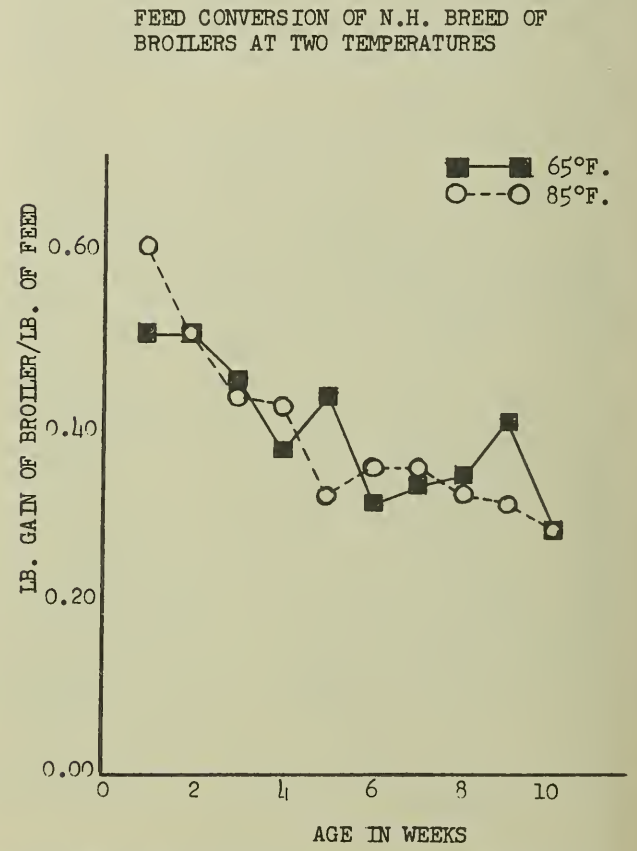

Figure 2.--Feed efficiency during first 10 weeks of growth from Beltsville, Md., poultry calorimeter data (9).

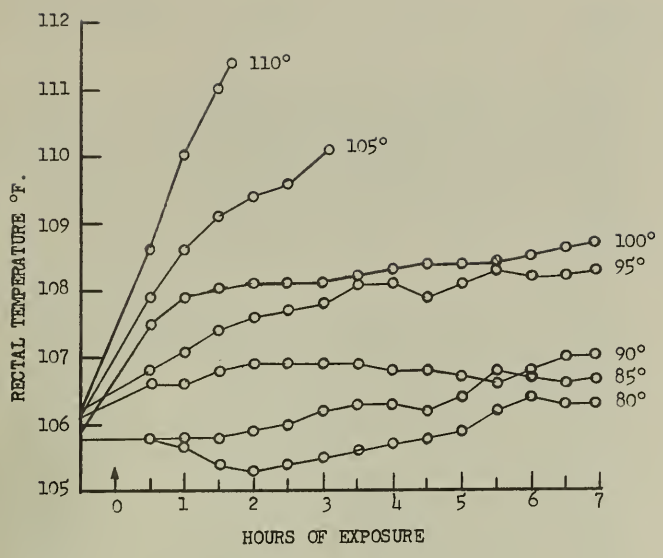

Figure 3.--Change in rectal temperatures with time upon exposure to various temperature levels and a relative humidity of 65 percent (15).

transferring four White Leghorn hens from a natural environment into various high temperature environments are shown. The variations in the initial level of rectal temperature and the erratic nature of the changes with time are characteristic of rectal temperature data of large animals as well as of poultry. Figure 3 indicates two important points. One, upon the change to high environmental conditions, rectal temperatures of chickens rose very rapidly. The duration of exposure at 105° F. was shortened because of the danger of heat prostration. This sudden rise may be expected when one considers that the thermal inertial of a small mass such as a chicken is much less than that of a large animal such as a cow. Two, at temperatures as low as 85° F., a rise in rectal temperatures occurred among these Leghorn hens. Generally, larger birds would be less heat tolerant (2, 5, 8) and younger birds would be more heat tolerant than older birds (10). The level of production and physiological condition of a chicken may also have a bearing on heat tolerance.

The data shown in Figure 3 were obtained at one relative humidity (65 percent). Exposures were also made at other relative humidities. Results showed that high humidity accentuated the harmful effects of high temperatures. This means that lowered ambient air temperatures at the expense of increased humidity (such as with evaporative coolers) may not always be beneficial. More extensive data are needed on the effects of humidity.

The effects of high temperatures on broiler growth (9) are shown in Figure 4. Here there is some indication of the harmful effects of an 85° F air temperature. After about 5 weeks of age the birds raised at 85° F gained at a slightly slower rate than those at 65° F.

Low levels of production, poor feed conversion efficiencies, heat stroke and chilling are direct indications of the harmful effects of poor thermal environments.

Disease may be considered an indirect result of a poor thermal environment. The multiplication and viability of pathogenic microorganisms in the air are functions of air temperature and humidity. Peak mortality among organisms has been reported to occur at 50 percent relative humidity (1). In addition, the concentration of disease transport vehicles such as water droplets and dust in the air are a function of the thermal environments. Added to this is the more subtle effect of lowered disease resistance brought on by "thermal stress". For instance, Van Ness (14) reported a marked increase in blue comb among chickens presented to the Florida laboratory 3 to 4 days after maximum temperatures exceeding 85° F. He also reported a marked increase in blue comb at five New England laboratories 2 days after maximum temperatures were 78° F. or higher.

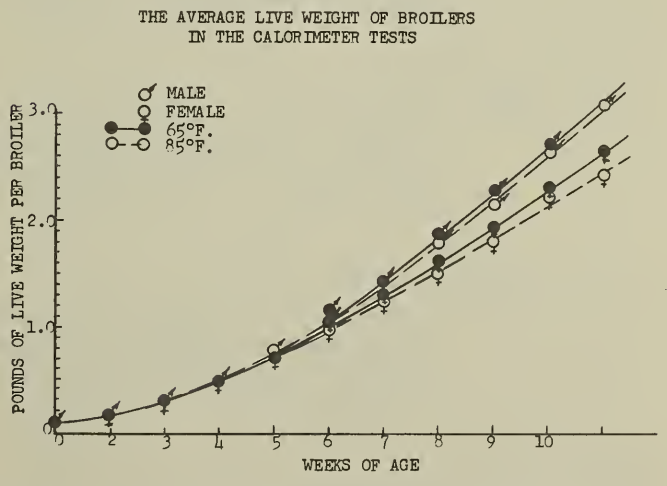

Figure 4.--Growth of New Hampshire Broilers at 65° and 85° F. Each treatment was initiated with 70 chickens. This number was periodically reduced to 35 chickens at 10 weeks. Data from Beltsville, Md., poultry calorimeter tests (9).

SUMMARY

The importance of thermal environments in poultry housing resolves itself into a proper balance between heat input and output. The heat transfer characteristics of birds have not been sufficiently determined to make theoretical calculations of all of the avenues of heat loss. Empirical evaluations of heat losses show that total heat output decreases with increasing air temperatures and that evaporative heat dissipation becomes more important with rising air temperatures. At 80° F., it accounts for more than 30 percent of the total heat loss.

The imbalance between heat input and output may be recognized by abnormal rectal temperatures. These generally occur between 85 and 95° F. but are also affected by humidity. High humidity accentuates the effect of high temperature.

Losses in production, lowered resistance to disease and even death have been correlated with thermal environments that cause abnormal rectal temperatures. Some of these effects may be noted even before rectal temperatures become abnormal.

Although the literature is fairly extensive on the subject of the effects of environment on poultry, most of it could be considered only exploratory. The complexity of the problem and the variations of each chicken, as well as those among chickens, are great. Extensive research with close control of all the environmental factors is required before sufficient quantitative data will be available to accurately evaluate the reaction of poultry to their thermal environments.

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CLIMATIC CONSIDERATIONS IN POULTRY HOUSE DESIGN

By Price Hobgood[1]

Your program indicates that I am to discuss climatic considerations in poultry house design. As you are aware, this is a rather broad subject which would permit me to discuss anything from the weather to building materials. Our good friend, Dr. Yeck, however, was quite alert and put some limits on the area. So I will discuss the subject from the standpoint of (1) the engineer's design problem, (2) methods that have been used in attempting to regulate environment for poultry, (3) what is happening in environmental control at the producer level, and (4) the need for knowledge concerning precise environmental requirements.

To give you some idea of the agricultural engineer's dilemma, I would like to take a quote from a typical letter received in our office last November. This is from the agricultural development director of a public utility company serving the high plains of Texas:

"I have a customer that is planning a new ten thousand bird laying house. The building will be 40 x 300 feet and the old boy wants complete environmental control. He is asking for information on forced air heating and cooling. I have read some of the reports that you have written on the work there at the college and am particularly interested in any special problems that may be encountered in designing the heating and cooling system for such an installation. The house will have the birds on the floor. My information is rather incomplete on specific requirements for poultry. Will you please furnish the information we need and our engineers can handle the design details."

To use a second illustration, last spring a man came into the office and wanted to

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know how to cool his laying houses for approximately 50,000 turkey hens. He was not wanting advice on whether or not to do it, but how. Frankly, he was a little impatient that we were hesitant in making recommendations. He did not seem to have visions of moisture, dust, colds, sanitation and, all the other problems that we could think of. Any way at his insistence, Dr. John Quisenberry of our Poultry Science Department and I made some recommendations with expressed fears and have held our breath at intervals ever since. So far we have heard nothing good or bad.

One section of our State produces a sizable volume of broilers. Some of the more aggressive producers use cooling equipment in the hot months and seem to get improved gains. However, on a few occasions such birds suffered high losses during transportation to the processor. As a result it is virtually impossible to sell broilers that have been produced under semi-controlled conditions if the buyer knows it.

Examples such as these cause engineers considerable apprehension. Let's look at this design problem a little more closely to see how nearly we can answer some of the questions that must be answered before more serviceable designs can be made.

What specific conditions are needed for the bird to be most effective in converting feed energy into useful meat and eggs? To answer this we must answer several specific questions within this area such as:

(a) What temperatures should be considered? Wilson, Ota, and others have done much very fine work in this area and have given us charts which tell us something of the effect of temperature upon the productive life of birds. Dr. Yeck has just presented valuable material in this area. What temperature range will give us the greatest return for our investment?

(b) What moisture content of the air is needed? Sometimes there appears to be conflicts on this point in the literature. Just what limits should the engineer use to make his design for moisture removal or addition.

(c) What tolerance should we consider for carbon dioxide, ammonia and other gases? Generally speaking some of these tolerances have been fairly well established. However, there seem to be questions among production people concerning the validity of some of the tolerances.

(d) What are the tolerances of air movement when combined with the above factors. There are many indications that layers are quite tolerant to higher velocities during the summer. At the same time there are indications that relatively lower air movement in cold weather will seriously affect production.

(e) What tolerances do we have to consider in terms of dust or fiber particles in the air? Many producers seem to be concerned about a house that is dusty.

(f) What are the effects of radiation sources on birds with a feather coat? We know that roof and wall temperatures of the light metal buildings often reach 150° or more in the summer and approach atmospheric temperatures in the winter months. A metal clad building may receive enough heat each hour during the middle of the day to potentially raise the inside air temperature 90° above the outside air unless insulation, shading or other means are provided to retard heat flow. Do the birds' feathers prevent heat gains and heat losses from such high or low temperature sources? People are quite sensitive to these sources of heat loss or heat gain.

(g) How much space is required per bird, based on some valid significant research. This item seems to be a never ending question which is highly debatable between producers.

(h) What quality and intensity of light is needed? Generally we realize that low intensities of light seem to be quite adequate. There seems to be little known concerning the effect of quality of light. All of these items would seem to be related to the stress under which the bird must produce.

At what level of environmental control will the bird be able to respond to justify additional cost through more efficient production, better health, and longer production life?

Many of the above questions have partial answers and some of them possibly have total answers if they can be interpreted in terms of sound engineering design.

A study of temperature and humidity conditions will usually indicate the potential control that may be obtained by evaporative methods. In a study of the temperatures that must be considered in our state, we found that we could normally expect from 300 to 600 hours annually in which the dry bulb temperature would exceed 90° F. We could

possibly expect an excess of 200 hours in most areas of the state in which it would exceed 95° F. This time would normally occur in 4 to 5 hour periods beginning around noon and lasting until 4 or 5 p.m. each day.

From studying temperature data in most of the south and southwestern states I would suspect that much of this area is subjected to similar temperature conditions. We also found that the temperatures may be expected to range from 105° F. along the Gulf Coast to as high as 115° in several areas of our State. These high temperatures normally occur during July and August. However, they may occur in June, July, August, or early September. Generally speaking, the temperatures that could be expected to occur frequently and to last for extended periods are almost constant across the entire state, ranging from 90° to 97° F. These temperatures are considerably above those found by Wilson, Ota, and others as to be desirable for poultry production. The mean relative humidity that can be expected ranges from 60 to 65 percent immediately on the Gulf Coast to 35 percent in the El Paso Region. This means that evaporative cooling will work in at least 50 percent of our state and probably in 80 percent if a highly efficient evaporative cooling system were used.

Our study also revealed that when the dry bulb temperature is excessively high the relative humidity invariably drops very radically and that we can expect to have a wet bulb depression of from 20° to 33° F. across most of the areas of our State. This indicates that we can succeed in lowering the dry bulb temperature by 70 to 80 percent of the wet bulb depression, and we can possibly get back into the range of 80° to 85° F. dry bulb on the hotter days.

Now let us study the conditions in a conventional house on a typical July day. The outside temperature rose above 100° F. at about 2 p.m. and remained that way until about 4 p.m. During this time the temperature in a check house was about 97° F. and that in a house with foggers approximately 95° F. During some periods of the day the temperature in the house with foggers was somewhat above that of the check house. The conventional evaporative coolers did a better job than the foggers. The house that was cooled with the pad and fan system did not exceed 80° F. on a typical hot day.

It was disappointing to note that the conventional evaporative cooler did not give the cooling effect that was obtained by the pad and fan system, since the pads were of the correct size with the proper air velocity and volume of air brought into the house was equal to that of the pad and fan system. Further study with a titanium tetrachloride smoke indicated that the difference in cooling between the conventional evaporative units and the pad and fan system was due to the turbulence and mixing of air within the house by the conventional coolers. The air was moved directly across the house and discharged by the pad and fan system. The fogging system had very little ventilation on still days. The smoke remained over very long periods of time and there seemed to be very little thermal movement within the house.

Many producers in our State have had heavy mortality during the summer, and as a result have installed some form of cooling equipment. Bozeman and Deacon working in the Dallas area with heavy breeds of hatching egg layers have had very definite success in obtaining a high degree of hatchage, fewer broken eggs, higher percentage of large and medium eggs, and generally better bird health. Their work was quite impressive with the heavy breeds. However, it was considerably less impressive with the light breeds. It was also less impressive with young birds.

I would like to summarize briefly our experience with environmental control. Four systems that have been used to produce some measure of environmental control are: (1) Conventional fans, (2) Evaporative cooling by conventional coolers or foggers, (3) Evaporative pad and exhaust fan, and (4) refrigerated air cooling and convector heating. Results from studies seem to indicate that: (1) Conventional evaporative coolers with air flow ranging from 0.4 to 0.6 air changes per minute will reduce mortality which is usually attributed to heat. However, there is no appreciable change in production and very little measurable change in ambient temperature within the building. (2) Ordinary fans with air flow rates ranging from 0.5 to 3.5 air changes per minute produce similar results. (3) Conventional evaporative coolers which provide 1.8 air changes per minute will significantly reduce the inside air temperature when exhaust fans are applied to the opposite side of the house. (4) Fans

should normally operate on and below the bird level rather than above it for cooling. When the fans are located over the birds, the hot air is drawn from near the roof onto the birds, thus raising the air temperature around the birds and increasing their discomfort. (5) Foggers will normally reduce mortality which is usually attributed to heat, but significant increase in production efficiency is difficult to obtain consistently and moisture problems quite often develop. (6) Most effective evaporative cooling was accomplished with an evaporative pad in combination with an exhaust fan when the air velocity through the pad was 150 feet per minute. The rate of flow of water was 1/3 to 1/2 gallons per minute per foot length of pad. The pad was of high density and was made of aspen wood shreds.

During our study with evaporative methods we also worked with one field installation with a refrigerated air cooling and forced air heating system in the house. This house was used for both broilers and layers for two years. In both cases the birds were on the floor. The system was a conventional $7\frac{1}{2}$ ton absorption system with ducts running down one side of the house. The walls and ceiling were insulated with full thick bats, except the lower 2 feet of the wall which was glass. This system did a very nice job of cooling and heating. However, the ammonia build-up within the house was extremely high and the birds' eyes showed considerable irritation. In addition it was almost impossible to keep filters in the system since there was considerable dust movement. This was a continuous problem as far as the producer was concerned. In two years we were never able to gather any satisfactory data because the filter problem made it almost impossible to maintain a uniform environment in the house. The system was designed for human comfort, and the dust, droppings, and excess moisture presented a problem that could not be handled by ordinary air conditioning design. As a result of this study, we are inclined to believe that it will be necessary to remove the droppings and practice a very rigid sanitation program within the house if conventional methods of refrigerated air cooling and hot air heating are to be successful. Otherwise it will be necessary to design a special system to fit the needs of this application.

The one thing that gives us most concern is that many of our producers have installed and are continuing to install various forms of partial environmental control, and we are not sure that this is a sound move. The first problem that arises is a system generally designed for summer cooling, which requires that the house is reasonably tight and that there be positive air movement in relatively large quantities a high percentage of the time. Yet when winter comes, these same houses (designed to provide summer comfort) present real sanitation, odor, temperature regulation, and moisture problems. We feel that there is a very great need for an evaluation of efforts that are being made to partially control environment in terms of bird health, productive life of bird, shell quality, breakage, mortality, egg size, feed conversion, and such other items that may contribute to a sound production practice.

The poultry industry has made great strides in breeding and feeding. As a result the entire consumer public has benefitted by having a higher quality meat at a lower price than was ever before possible in our history. Many of the other poultry production practices have not kept pace with these two areas. As a result much effort should be put forth to determine precise environmental requirements and means by which these requirements can be met. Generally, the things that have been done thus far have been accomplished by possibly one, two, or three disciplines. However, I believe our greatest progress will be made when a team of physiologists, geneticists, economists, engineers, veterinarians and other disciplines join the production man to attack the problems.

SECTION VI--SUMMARY

PROGRAM SUMMARY

By W. C. Patterson[1]

In attempting to summarize this program, I am reminded of the verse,

"Big flees have little fleas
Upon their backs to bite 'em,
And little fleas have lesser fleas,
And so, ad infinitum."

Dr. Hegner's problem of the pestiferous fleas and the protozoa which inhabit their little bellies seems no less complex than some of those problems we have been discussing during the past few days. For, as Dr. Hegner said, to understand how the other half lives, you must first understand the protozoa, then the partnership nature imposes on the beasts and fowls, but this is not enough. What you eat determines the character of your unwelcome guests and where you live decides what bugs shall occupy your interior. And this is exactly what we have been talking about--the disease agent and its host, plus a few complicating disease agents, plus other external and internal considerations.

As Dr. Byerly said on Monday, the first step in problem-oriented or applied research is to define the problem. This we have attempted to do. What is causing the high condemnation loss in broilers? What disease, environmental, and management factors affect poultry health? Dr. Schmittle has reported the results of field studies of condemnation losses in Georgia. The results show that disease, particularly respiratory diseases in the presence of adverse environmental influences, directly affect the condemnation rate of broilers. The respiratory diseases, in their order of importance are: chronic respiratory disease (PPLO infection), infectious bronchitis, and Newcastle disease. Respiratory diseases are the most important factors related to condemnations, and the combination of adverse environmental conditions with the presence of disease resulted in the highest condemnations. The qualities which lead to good record keeping (management) also produce good poultry and less condemnation.

A survey in the Delmarva area on factors of management and disease control which influence the condemnation rate was reported by Dr. Cover. It showed that (1) the use of large numbers of chicks around one stove tended to increase the rate of condemnation, (2) condemnations were less in the narrow houses than the wide houses, (3) condemnations in houses using individual gas stoves were higher than in houses using oil stoves, (4) condemnation in houses having solid partitions between pens was less than in houses having open partitions, (5) those management practices associated with good sanitation reduced the condemnation rate and, (6) the average condemnation rate was lower in flocks receiving the recommended vaccination program.

In reporting on a program inaugurated in September 1960 in Louisiana, Dr. Dixon

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reports that condemnation rates have been decreasing and the outlook is that they will continue to decrease. Condemnations in the current program have been charged as follows: on post mortem--grower - 56 percent, processing plant - 19 percent, breeder hen - 17.5 percent, hatchery - 4 percent, catchers and haulers - 2 percent, and inspection - 1.5 percent. On ante mortem--hauler - 86 percent, processing plant - 9 percent, and breeder hen - 5 percent. Condemnation reductions are attributed to a better job being done by the grower and stabilization of the inspection service.

Dr. Edgar, in an Alabama survey, listed the following production factors as increasing condemnation: large flocks and wide and long houses, inexperienced growers, overcrowding under brooders, respiratory disease outbreaks, persistence of moderate infections of roundworms or coccidiosis, limited feeding of birds, and poor feed conversion. A study of birds from those condemned at processing reveal disagreement with the inspection service approximately 50% of the time. Variation in inspection between plants and inspectors seemed excessive. The percentage of agreement improved with time. The greatest cause of condemnation was a generalized inflammatory process due to CRD.

So much about surveys: What about the diseases involved in condemnations? Dr. Van Roekel has made a current evaluation of chronic respiratory disease which is a major disease problem among out chicken and turkey industries. While the etiology of the disease is known, our knowledge of possible serotypes and their host parasite relationship is incomplete. Egg transmission of the disease appears to be the primary mode of the dissemination of the agent. Medication for the prevention and control of CRD outbreaks has yielded variable results. Recently the effectiveness of certain antibiotics has been increased through potentiation with low calcium diets and terephthalic acid. Control and prevention of the disease through immunization remain virtually unexplored. Lastly, the full significance of CRD in the aerosaculitis syndrome cannot be established unequivocally until stock free of CRD can be produced consistently in a clean environment.

Dr. Grumbles, in reviewing the role of the principal acute viral respiratory diseases in the chronic respiratory disease--air sac syndrome, emphasized the fact that surprisingly few experiments have been designed to show the role of infectious bronchitis, Newcastle disease, or laryngotracheitis in CRD-air sac infections. It has been shown conclusively that acute viral respiratory infections may: (1) activate latent mycoplasma infections, (2) "trigger" field outbreaks of "air-sac" disease, (3) increase impairment of performance and (4) cause much more severe symptoms and lesions in birds with the CRD-air sac syndrome.

Among the more common lesions of birds rejected for wholesomeness are pericarditis and perihepatitis. From the pericardial sacs and livers Escherichia coli can be isolated. Dr. Gross reported experimental work which showed that birds having a respiratory disease due to PPLO, infectious bronchitis, or Newcastle disease alone or in combination, became susceptible to E. coli administration in an aerosol.

The most important agent is PPLO which when given alone into the air sacs of chickens renders them susceptible to E. coli invasion for from 8 to 40 days. Vaccine viruses are almost as effective as field strains in making PPLO-infected birds susceptible to E. coli.

Dr. Burmester, in discussing the avian leukosis complex problem, emphasized the fact that leukosis continues to be a constant but unspectacular problem and, in fact, is increasing in many areas where losses due to CRD have remained almost unchanged. Research needs for this disease complex were outlined.

The importance of prompt isolation, identification, and typing of Salmonella organisms when present in poultry, their feed, or their environment, and the immediate reporting of these findings to the responsible disease officials was stressed by Dr. Stover in his discussion on the control of Salmonella infections.

Dr. Reid, in outlining needs for the control of coccidiosis, suggested the following fields: (1) a continued search for cheaper and better coccidiostats, (2) search for improving methods of application and efficiency for planned immunization programs, (3) rapid method for detecting flock immunity, (4) establishment of a repository of coccidial species and strains.

Dr. Cover stressed that the value of any medicament in treating cases of air sac will be measured in its ability to

control secondary infection. While there has been considerable research into methods of preventing air sac infections, there is little encouraging information. At the present time, an antibiotic egg dipping technique shows promise.

Dr. Levine, in considering immunizing procedures in relation to air sac infections, considers the basic problem to be the mechanism of the initiation of CRD outbreaks in young birds in the field. The most apparent predisposing factor to CRD in the field is infectious bronchitis and Newcastle disease vaccination.

As an assist to better understand how disease pathogens act on air sac tissues, Dr. Lucas's excellent presentation described the location of air sacs and gave basic information on the histology of these organs.

What about management considerations in relation to poultry health? Dr. Hutt said there are already enough indications of genetic resistance to suggest that with appropriate selection, the proportion of birds able to cope with normal exposure to the organisms concerned could be raised significantly and losses reduced proportionately. Procedures for doing this have been outlined. The task is one for teamwork by pathologists, nutritionists, physiologists, and geneticists.

Dr. Hill's work shows that nutrition can influence disease. The extent or direction of this work is not yet predictable, but certainly a field worth exploration.

Dr. Sturkie, in discussing physiological response to management, described the effect of season and temperature on cardiac output and blood pressure in birds, and the relationship of the level of blood pressures to mortality and resistance to physical stresses. Contrary to humans, low blood pressure kills more chickens than high blood pressure. It is suggested that high blood pressure birds are better able to withstand disease exposure and stress. Hypertensive birds had better survival to low temperatures and fatigue.

To be of value in reducing broiler condemnations, husbandry practices must be part of a coordinated program of environmental control and disease prevention. Dr. Parkhurst expressed the importance of broiler producers putting into practice available information on sanitation, good management, and disease prevention.

Now for engineering considerations--

Dr. Hinshaw stated that the basic principle for economic rearing of birds is the use of disease-free flocks. If maintenance of disease-free flocks is not possible, then "convalescent rearing" becomes important and engineering research on housing and environmental factors as they affect the health of the bird becomes even more important.

Dr. Brown, in reviewing engineering factors related to poultry health, stressed the three "R's"--removing disease, relieving condition, and recovery from disease. Engineering has much to offer in each of these considerations.

Dr. Horton's paper discussed several aspects of the role of air in the transmission of disease. Air, being a vehicle of infection, must be considered in controlling the spread of microorganisms. Dr. Decker's paper reviewed the efficiencies of various air cleaning equipment in removing microbiological particles 1 to 5 microns in size. A primary consideration of filtration would be, does the need justify the expense? His conclusion: where poultry houses use forced ventilation systems, and there are 10 or more changes per hour, with no air being recirculated, it is unnecessary to filter air if the exhaust ducts are not located near the inlet ducts.

Dr. Yeck reviewed for us factors involved with heat exchange between poultry and their environment as well as some of the reported effects of thermal environments on poultry. Loss of production, lowered resistance to disease, and eventually death, have occurred as the balance between heat input and output was lost.

Poultry house design must include not only structure, but also means by which temperature, humidity, ventilation rate, and dust can be controlled to a degree required for bird health and economic production. Professor Hobgood, in considering these problems, has just told us that lack of technical information is primarily in the area of exact tolerances of poultry to the various elements of environment.

Well, there you have it! The problem has been defined and many suggestions have been made for resolution. One thing seems to be missing. The magic pill! The chartreuse capsule or magic nostrum which will solve all our problems. Unfortunately, there is none!

We have a tendency to be impatient with research for there are so many questions which need answering, and I would have to agree, for I, too, get

impatient when I can fly from here to New York in 50 minutes and have to spend an additional hour in the air before landing, because of crowded airports or bad weather. For the hard fact is that even the finest of airplanes is not worth much if the weather is too soupy for flying or the runways too crowded for landing.

What I'm saying is that we are all in this thing together--producer, processor, regulatory official, research worker, extension specialist. Dick Hanson has said that our problems range from viruses to people. Research might cope with the viruses, but we need two-way communication to open both ends of our program and prevent much misunderstanding. Julius Bishop emphasized the importance of having information made available for practical use in the field. As he said, the poultry industry moves at such a rapid pace, it is difficult for the producer to stay abreast of developments.

Dr. Byerly has said we need both basic and applied research in three directions: (1) on environmental control to provide better growing conditions in the absence, in the prevention of transmission, and in the presence of disease; (2) on control of disease through immunization, genetic resistance, prophylaxis, and therapy; and finally, (3) on disease eradication. Research in these three areas should be concurrent and coordinated.

An evaluation of our research needs for poultry health clearly shows the value of a coordinated program to make the most effective use of the efforts available. Public and private research--federal, state, industrial--should coordinate their efforts as closely as possible in order to make faster progress. Close coordination in planning research programs and in exchanging results could help to expand the total value of the time, money, manpower, and facilities available throughout the nation.

It is hoped that this symposium has stimulated our thinking of the interrelation of disease, environmental, and management factors related to poultry health. Beyond this, it is hoped that we have considered our dependence upon each other and the importance of cooperating to solve a common problem.

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